DISSERTATION ON

A STUDY ON PROGNOSTIC SIGNIFICANCE OF WHITE BLOOD CELL COUNT AND BLOOD GLUCOSE LEVELS AT ADMISSION IN ST ELEVATION MYOCARDIAL INFARCTION

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CERTIFICATE

This is to certify that this dissertation entitled

"A STUDY ON PROGNOSTIC SIGNIFICANCE OF WHITE BLOOD CELL COUNT AND BLOOD GLUCOSE LEVELS AT ADMISSION IN ST ELEVATION MYOCARDIAL INFARCTION"

is the bonafide original work of **Dr.VINOTH KANNAN .R** in partial fulfilment of the requirements for M.D Branch -I (General Medicine) Examination of the TamilnaduDr. M.G.R. Medical University to be held in APRIL - 2015. The period of study was from January– 2014 - August 2014.

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DECLARATION

I, **Dr.VINOTH KANNAN.R** , solemnly declare that the dissertation titled DISSERTATION ON **"A STUDY ON PROGNOSTIC SIGNIFICANCE OF WHITE BLOOD CELL COUNT AND BLOOD GLUCOSE LEVELS AT ADMISSION IN ST ELEVATION MYOCARDIAL INFARCTION"** is a bonafide workdone by me at Thanjavur Medical College, Thanjavur during Januuary 2014 – August 2014 under the guidance and supervision of **Prof.Dr.C.GANESAN, M.D.,** Unit Chief M-4, Thanjavur Medical College, Thanjavur.

This dissertation is submitted to TamilnaduDr. M.G.R Medical University towards partial fulfilment of requirement for the award of **M.D. degree (Branch -I) in General Medicine**.

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CONTENTS

ABSTRACT

Background and objective:

Admission white blood cell (WBC) count and Random Blood Sugar glucose (RBS) have been associated with adverse outcomes after acute myocardial infarction (AMI). This study investigated the combined effect of WBC count and Random Blood Sugar on predicting in-hospital mortality in patients with acute myocardial infarction.

Material and methods:

80 consecutive patients presenting with acute ST elevation myocardial infarction admitted in IMCU/ICCU Thanjavur Medical College Hospital from January 2014 to August 2014 were studied. Patients satisfied the inclusion criteria underwent detailed history and clinical examination. WBC count and RBS were measured at the time of hospital admission and the patients were stratified into 3 groups (low, medium, and high) based on their WBC count and blood glucose.

Results :

Patients with a high RBS had a four fold increase in in-hospital mortality and the patients with a high WBC count had a ten fold increase in mortality when compared with those with normal values. When a combination of different strata for each variable was analyzed, a stepwise increase in mortality was seen. Patients with high WBC count and normal RBS level or with a normal WBC count and

high RBS values had intermediate risk. Multivariate analysis was performed to assess the predictors for in-hospital mortality using WBC count and RBS level as continuous variables and it showed these two were independently associated with in-hospital mortality.

Conclusion: W.B.C count and plasma glucose level at admission has a prognostic importance in predicting in-hospital mortality in acute myocardial infarction.

Keywords: MI-Myocardial infarction **; W**.B.C – White blood cell ; RBS- Random blood sugar.

INTRODUCTION:

Ischemic heart disease is one of the most common non-communicable disease causing mortality, morbidity and incurs great economic burden both in the developed and developing countries. Its incidence is on the rise due to sedentary lifestyle and increasing urbanization. In the next ten years it is going to be the most common cause of death worldwide.

It has been found in the recent years that the non- diabetic patients with acute myocardial infarction present with hyperglycemia, glycosuria and insulin resistance due to neurohumoral activation. Even though it is a physiological adaptive response; several studies in the recent past had shown that the high blood sugar values were associated with poor outcome in these patients.

Several epidemiological studies have demonstrated the elevated levels of highly sensitive C- reactive protein, Interleukin-1b , Interleukin 6 and tumour necrosis factor in ischemic heart disease patients and their levels peak during the event of acute coronary syndrome.

The routine traditional risk factors like advanced age, smoking, diabetes, hypertension, dyslipidemia has been paid much attention and the role of inflammatory markers and stress hyperglycemia were neglected.

The total white blood cell count and the blood sugar values are frequently found elevated during the time of admission in acute coronary syndromes. Hyperglycemia may be due to pre-existing diabetes or due to simple stress hyperglycemia. The leukocytes are elevated physiologically as an adaptive response that is essential for wound healing and repair.

Any exaggerated physiological response may always have some pathological consequences that is applicable here also. Hyperglycemia can produce free radical injury of myocytes, prevent blood flow in the occluded coronary vessels during reperfusion , dysfunction of vascular endothelium. Similarly elevated leukocyte count may promote coagulation, prevents micro vascular reflow, exaggerates inflammation and myocyte injury.

Hence an elevated levels of blood sugar levels and WBC count at the time of event in acute coronary syndrome usually have poor prognosis in terms of high in hospital mortality and decreased ejection fraction. The independent association of these two variables with prognosis have been evaluated in many other studies previously.

Determination of blood sugar and WBC count is a simple procedure, requires no expertise, is inexpensive and importantly, it is a correctable factor. Based on conflicting literature and because of lack of similar studies conducted in our region, this study aims at exploring the association between the admission glycemic status and and leukocyte

count with in- hospital mortality in acute ST elevation myocardial infarction patients. Hence this is a novel study that assess the combined effect of blood glucose and WBC count in predicting the complications and mortality of ST elevation myocardial infarction patients.

AIMS AND OBJECTIVES:

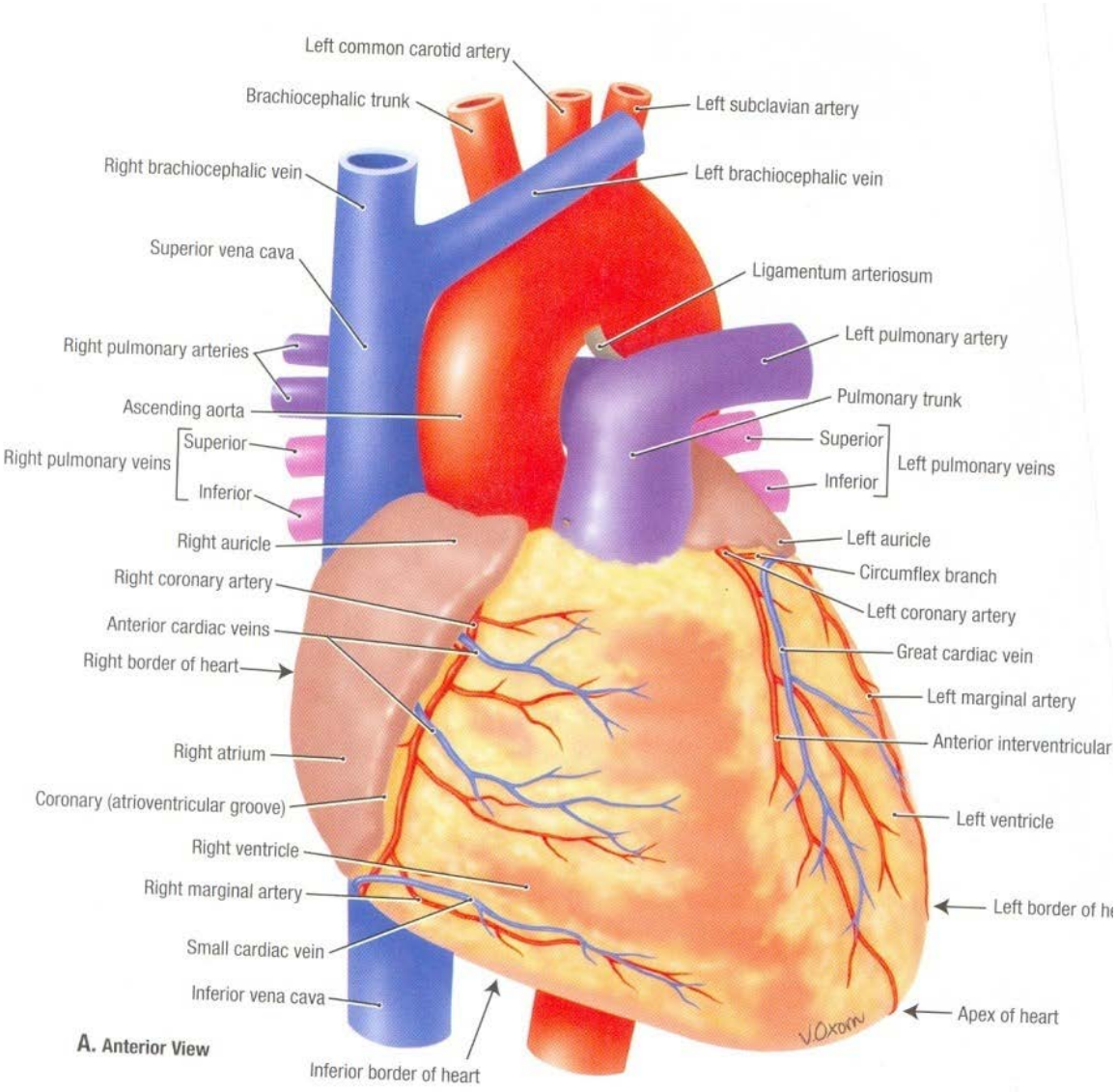
The study is designed to assess;

- the prognostic significance of white blood cell count and plasma glucose levels at admission in acute ST elevation myocardial infarction within 48 hours in terms of in –hospital mortality,
- the significance of white blood cell count and plasma glucose levels on predicting the cardiac functioning capacity in terms of Ejection Fraction during the hospital stay.

REVIEW OF LITERATURE:

ANATOMY OF HEART:

Fig 1: HEART AND CORONARY VESSELS- ANTEROR VIEW



- The right cardiac border formed mainly by the right atrium is convex and in line with the superior vena cava an inferior vena cava.

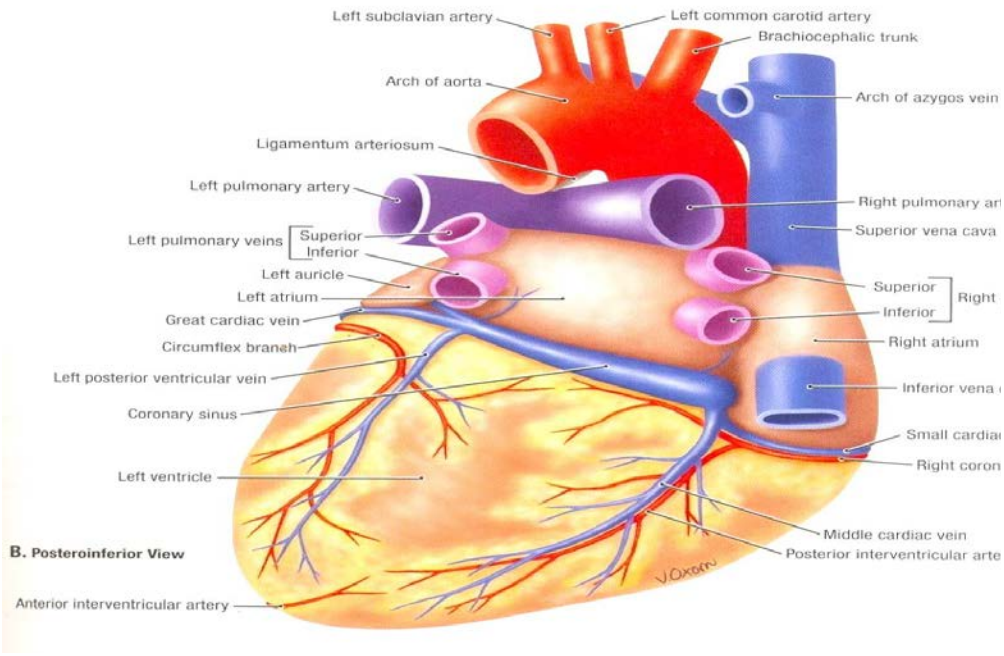
- The inferior border is formed mostly by the right ventricle and a small portion of the left ventricle.
- The left border is formed predominantly by the left ventricle and a small portion of the left auricular appendages.
- The main pulmonary artery bifurcates into a right pulmonary artery that passes under the aortic arch and left pulmonary artery.
- The right and left pulmonary veins open into the posterior wall of left atrium as four in number.

CORONARY ARTERIAL SUPPLY:

- The right and left coronary arteries : major blood vessels are the branches from ascending aorta in anterior and posterior sinuses .
- The Right coronary artery travels in the coronary sulcus to reach the posterior surface of the heart, where it anastomoses with the circumflex branch of the left coronary artery. It gives off the 1)Sinoatrial (SA) nodal artery that supplies the right atrium and SA node; 2)Marginal branch supplying most of the anterior wall of the right ventricle, 3)Atrioventricular nodal artery near the posterior border of the interventricular septum, and 4)posterior interventricular artery in the interventricular groove that anastomoses with the anterior interventricular artery.
- Left coronary artery gives the following branches 1)Circumflex

artery that passes posteriorly to anastomose with right coronary artery and 2) Anterior descending branch in the interventricular groove.The interventricular septum receives its blood supply from septal branches of the both descending arteries:-the anterior 2/3rd from the left coronary and the posterior 1/3 rd from the right coronary artery.

Fig 2: HEART AND CORONARY VESSELS- POSTERO INFERIOR VIEW



- The term 'dominant system ' is usually to denote the predominance of the right or left coronary in supplying the heart. It is decided by the artery which gives rise to posterior interventricular branch ; in 70- 75% of the individuals it is usually the left coronary artery. This posterior interventricular artery supplies the posterior two thirds of the interventricular septum.
- Coronary arteries are end arteries during adult life. Collateral will develop only in stages of chronic hypoxia. But the situation is reverse during fetal life in that they will have rich collateral network.

Control of Coronary Blood Flow:

1. Local metabolism

2. Nervous control

1) Local metbolism of myocardium:

The factors responseible are:

a) **Oxygen Demand**:

A major factor of all: As oxygen extraction is near complete in resting state only, increase in oxygen demand has to be met with by increasing the blood flow. This is achieved by the following mechanisms:

1. **Vasodilator Theory**: Anoxia will liberate many vasodilator materials from myocardial cells which increase the blood flow:

i) Adenosine

ii) Potassium ion

iii) Hydrogen ion

iv) Carbon dioxide

v) Bradykinin and possibly

vi) Prostaglindins

2. **Arterial Smooth Muscle Relaxation Theory**: Decrease in oxygen supply leads to anoxia of coronary arterial smooth muscle cells, which loses their tone thus getting the artery dilated.

Factors that determine the oxygen consumption are:

i) Increased arterial pressure, increases the work load and hence tension.

ii) Dilatation of the heart increases the tension development in myocardium to pump the blood according to Laplace law, which states that tension required to generate a given pressure increases in proportion to the diameter of the heart.

iii) Other factors which increases the oxygen consumption like stimulation of the heart by epinephrine and norepinephrine, thyroxine, digitalis, calcium ions, increased temperature of heart, will increase the oxygen consumption.

iv) Reactive hyperemia: Anoxia brings about increase flow because of coronary dilatation after a brief period of coronary occlusion.

Nervous Control

a) **Indirect**: Sympathetics increases the heart rate and contractility, through the local metabolic mechanisms, and hence increases the coronary flow. Parasympathetics decrease the heart rate and depresses the myocardium and hence brings about coronary constriction.

b) **Direct Effect:**

Parasympathetics: As the vagal supply to ventricles is negligible, except for slight dilatation which may occur, there is no effect of its stimulation.

Sympathetics: Epinephrine and norepinephrine through their receptors in coronary vessels usually bring about vasoconstriction or no change. When alpha effect dominates, severe constriction occurs which may bring about anginal attack.

DEFINITION OF MYOCARDIAL INFARCTION:

In 2001 the American College of Cardiology issued a joint recommendation for the diagnosis of Acute MI replacing the old version of World Health Organization definition. The typical rise and fall of cardiac markers like troponin or creatinine kinase myocardial band subunit in addition to at least any one of the below finding:

- Symptoms typically suggestive of myocardial ischemia like prolonged angina, palpitation, diaphoresis.
- Electrocardiographic changes indicating myocardial ischemia (ST-segment depression or elevation)
- New onset deep pathological Q waves

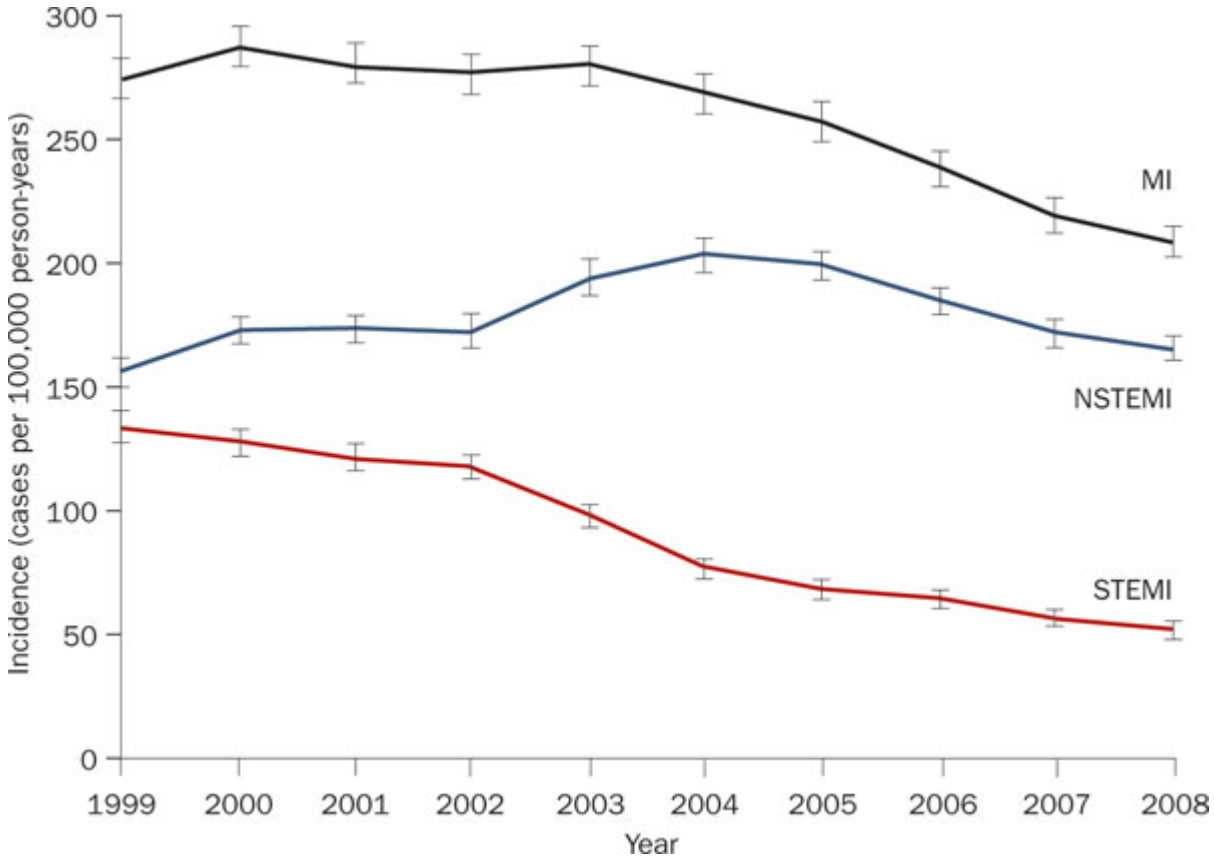
Pathologic findings of AMI at autopsy are also considered diagnostic[(1)]. Additionally, any patient presenting with a clinical picture consistent with myocardial infarction with a combination of new onset left bundle branch block can be be considered as STEMI. Detectable elevations of markers like troponin and CK-MB may not be apparent in those presenting within the first few hours (4- 6hours)of onset of the event. Based on these guidelines , the distinction between UA and NSTEMI may not be possible at the time of initial evaluation and require serial measurements of cardiac biomarkers.

MYOCARDIAL INFARCTION

EPIDEMIOLOGY

Ischemic heart disease (IHD) is a condition in which there is an mismatch between the available blood supply of myocardium and oxygen demand of that area. It is caused by either obstruction to the blood flow in a coronary vessel or increased metabolic demands of myocardium like cardiac chamber hypertrophy. Myocardial infarction is the ischemic necrosis of myocardium.

The most common etiology of MI is atherosclerotic narrowing or obstruction of an epicardial coronary artery that is sufficient to cause a regional reduction in myocardial blood flow and inadequate perfusion of the myocardium in that area.



IHD causes more mortality and morbidity throughout the world and also makes a great

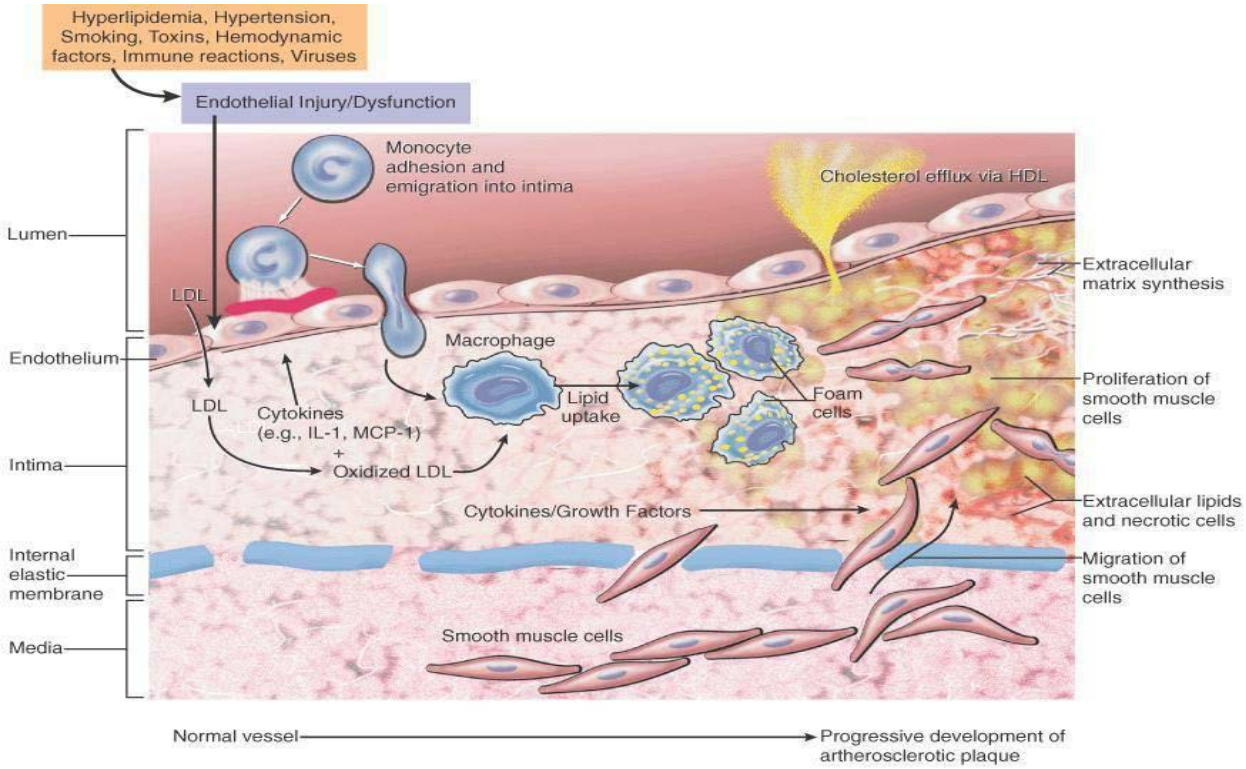
economic burden over the government. In the United States nearly 1.5 crore persons have IHD,among which more than 70 lakhs people have angina pectoris, and >7 million have sustained a attack of myocardial infarction in his lifetime. The early mortality i.e death within one month of acute myocardial infarction is nearly 35- 40%, with majority of deaths occurring before the affected individual reaches the hospital Despite these sobering data, it is worth noting that epidemiologic data show a decline in the rate of deaths due to IHD, about half of which is attributable to treatments and half to prevention by risk factor modification[2].

Obesity including metabolic syndrome , genetic factors like familial hyperlipidemias, high fat/ calory rich diet, sedentary life habits, insulin resistance, and type 2 diabetes mellitus are imajor risk factors for IHD. South east Asian countries are the population with high susceptibility.

PATHOPHYSIOLOGY:

Atherosclerotic plaque usually contains a central lipid core surrounded by a fibrous cap. Inflammation in the plaque recruits white blood cells mainly monocytes from the blood stream. Enzymes released from these monocytes cause oxidation of LDL . chemotactic factors from these leukocytes recruits more and more cells from the blood that intensifies the inflammation. Growth factors from these cells causes proliferation of smooth muscle cells in the media. Finally the metalloproteases released causes thinning , erosion and rupture of the fibrous cap that completely occludes the blood vessel lumen[3].

Fig 3: MECHANISM OF ATHEROMATOUS PLAQUE FORMATION:



Major Risk Factors for Atherosclerosis

NONMODIFIABLE

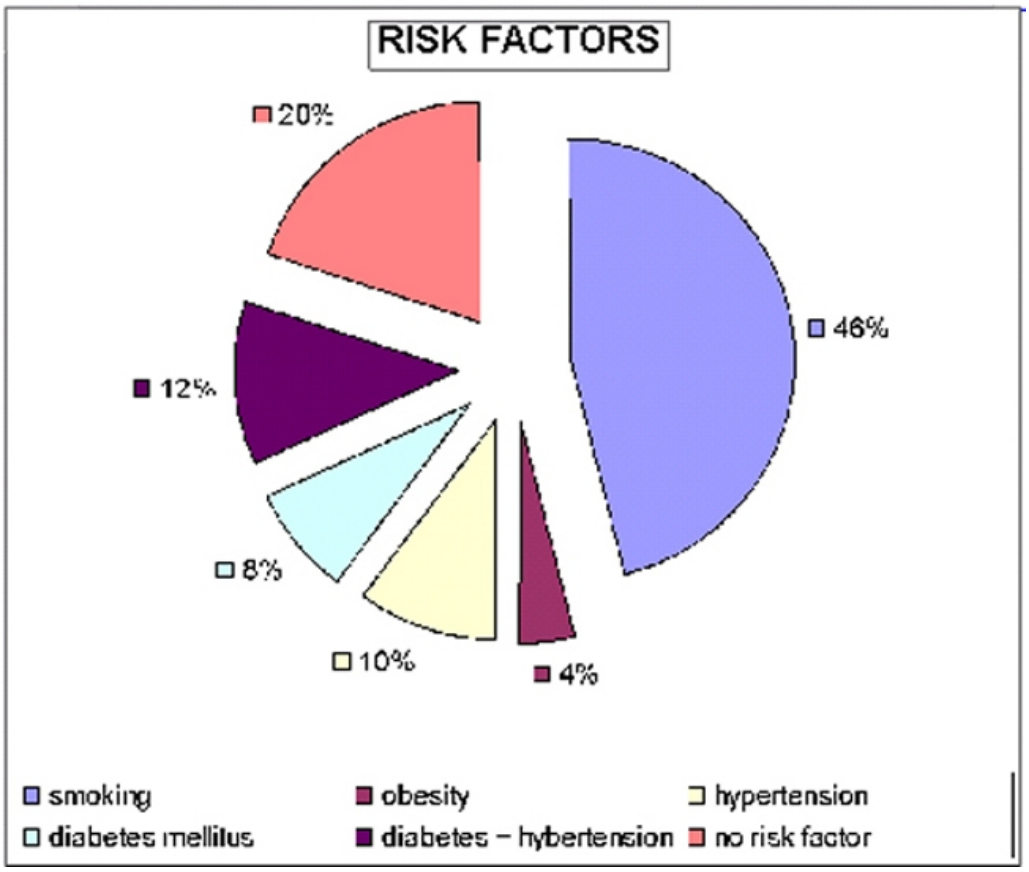
Advance age that includes men more than 45 years and women more than 55 years

Male gender

Positive Family history of early onset CAD (at age less than 55 years)

Genetic abnormalities

MODIFIABLE



Hyperlipidemia

Total cholesterol > 150mg%

Triglycerides > 150 mgs%

LDL cholesterol > 100 mg%

APO – b lipoproteins >100 mg %

HDL cholesterol < 40 mg% males, < 50 mg% females.

Hypertension

Cigarette smoking

Diabetes Mellitus

C-reactive protein

Apple obesity or BMI > 22

Homocysteine > 10 micro mol/lit

STAGES OF ATHEROSCLEROSIS:

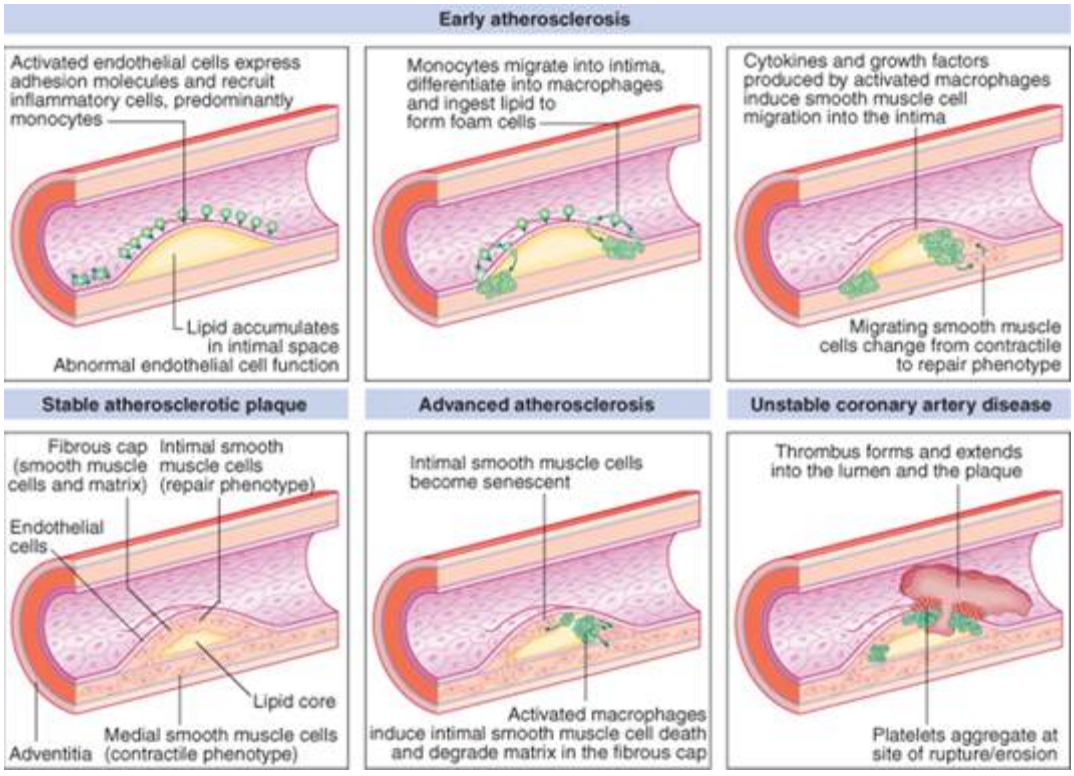
Early atherosclerosis

Subtle atherosclerosis

Advance atherosclerosis

Unstable coronary plaque[5]

Fig 4: STAGES OF ATHEROSCLEROSIS:



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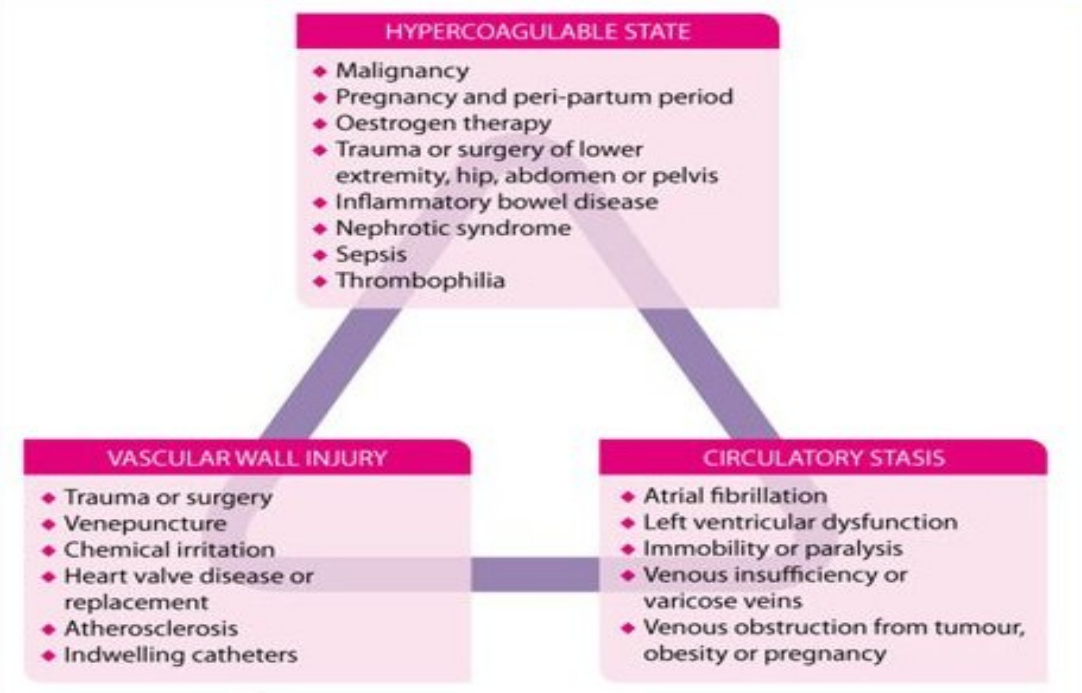
VIRCHOWs TRIAD OF THROMBUS FORMATION:

Virchows triad that is proposed for any vascular thrombus formation is applicable for

coronary thrombosis also. It includes hypercoagulable states as in familial thrombophilias like homocystinemia; abnormal vascular endothelium as in atherosclerosis; and vascular stasis in condition such as atrial fibrillation and severe ventricular dysfunction[4].

Fig 5: VIRCOWs TRIAD:



Causes of Acute MI:

CORONARY ATHEROSCLEROSIS:Almost all MI's result from coronary atherosclerosis

NON ATHEROSCLEROTIC CORONARY ARTERY DISEASE[6]

Arteritis as in collagen vascular disorders

Traumatic injury of the coronaries

Intimal Proliferative disease

- Mucopolysaccharidoses

- Homocysteinemia
- Fabry disease
- Amyloidosis
- Juvenile intimal sclerosis
- Oral contraceptive pills

Luminal narrowing

- prinzmetals angina ; spasm without thrombus
- Nitroglycerin withdrawal
- Aortic Dissection

Coronary artery Embolism

- Acute bacterial endocarditis
- Libman sachs and marantic endocarditis
- left ventricular stasis/ thrombus
- Cardiac tumours
- Post procedures like CABG and PTCA

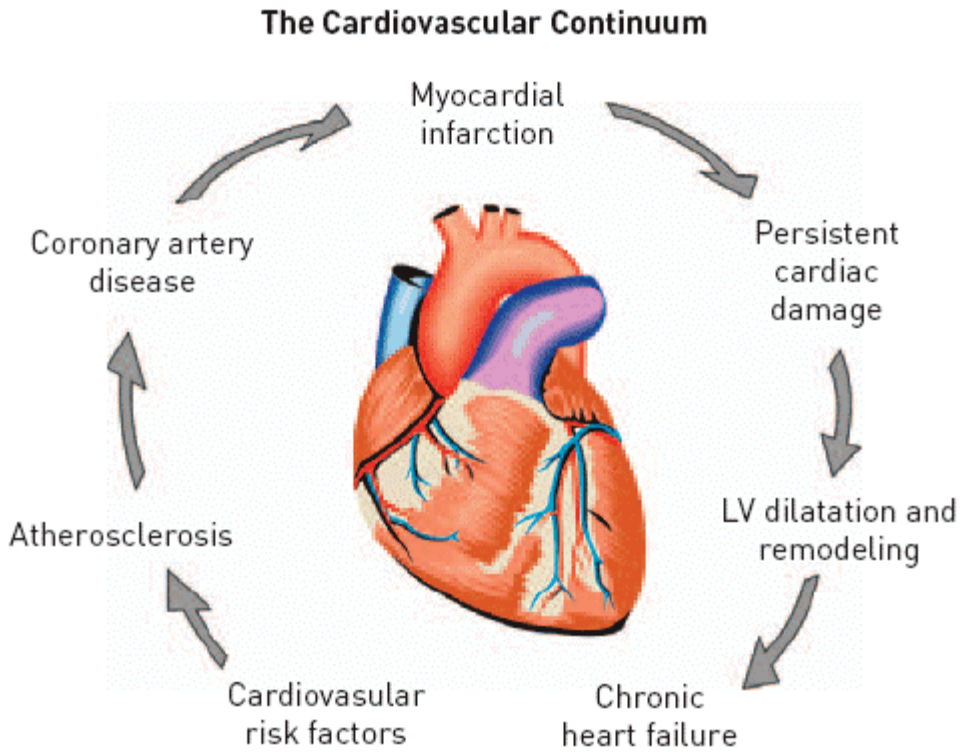
Congenital anomalies

- ALCAPA

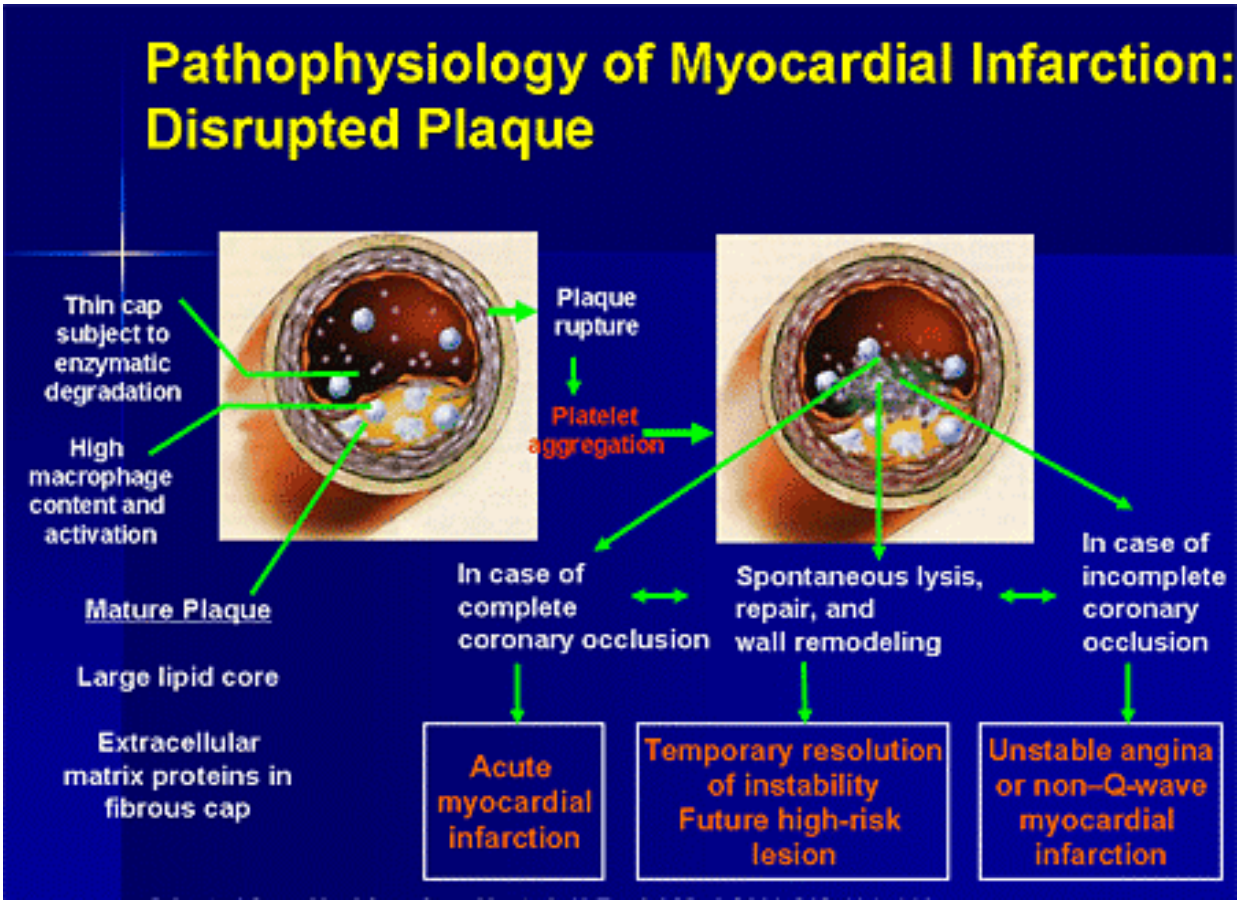
Myocardial oxygen demand-supply mismatch

- Aortic stenosis,
- Cardiac chamber hypertrophy
- Severe anaemia
- Pagets disease
- Cardiogenic shock

NATURAL HISTORY OF MYOCADIAL INFARCTION:



PATHOPHYSIOLOGY OF ACUTE CORONARY SYNDROME:



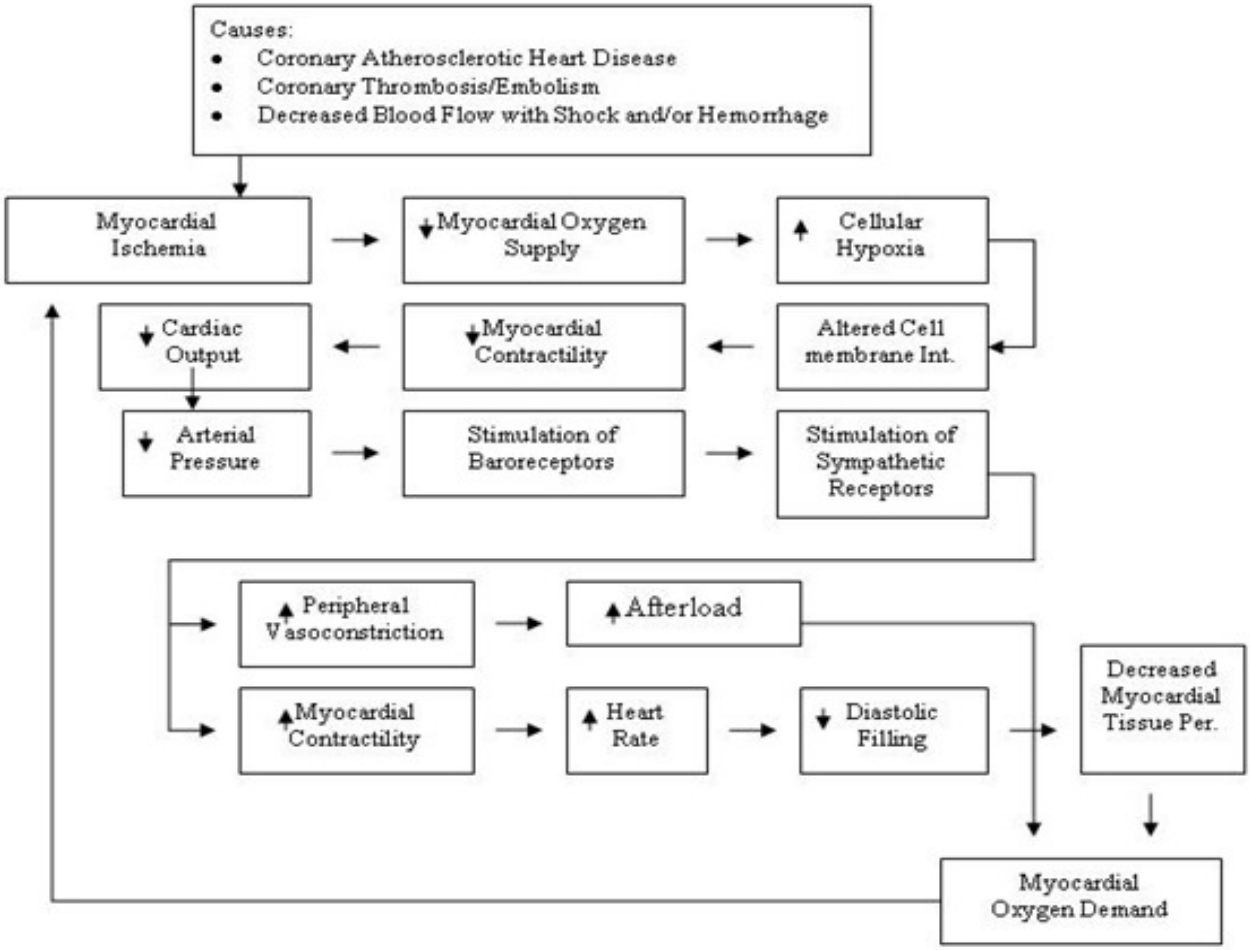
The clinical picture of acute coronary syndrome may vary depending upon the fate of the ruptured atherosclerotic plaque,[7]

- Complete occlusion results in transmural (ST elevation) MI
- Partial occlusion results in unstable angina / non ST elevation MI
- Sometimes spontaneous resolution can occur

HAEMODYNAMIC ALTERATIONS IN MI:

Myocardial infarction results in decreased contractility of heart and there by reduced stroke volume that stimulates the baroreceptors to increase the

sympathetic outflow. This sympathetic surge increases the heart rate, increases afterload, and decreases the diastolic filling that results in a vicious cycle.



CLINICAL FEATURES:

"Crushing" pain that lasts more than 15 minutes to hours

- *Clinical Features:*
  - Acute distress
  - Pallor
  - Perspiration and cold moist skin
  - Nausea, Vomiting, and Abdominal bloating
- *Vital Signs:*
  - Rapid Thready Pulse; can frequently be Irregular
  - DECREASED or DROPPING Blood Pressure
  - Respiration is shallow and the patient experiences dyspnea

OTHER SYMPTOMS

Diaphoresis, nausea, and vomiting , cold peripheries, delirium, giddiness, abdominal discomfort, unexplained syncopal attacks[8].

SIGNS:

Rapid thready (low volume) pulse

Elevated blood pressure due to pain

Low blood pressure due to cardiogenic shock

Elevated jugular venous pulse

Peripheral and central cyanosis

Febrile because of ongoing inflammation

Tachypnoea in left ventricular dysfunction

Tachycardia or bradycardia(inferior wall MI)

Lung crepitations in pulmonary edema

Muffled heart sounds especially S1

Systolic murmur due to ventricular septal rupture or papillary muscle rupture

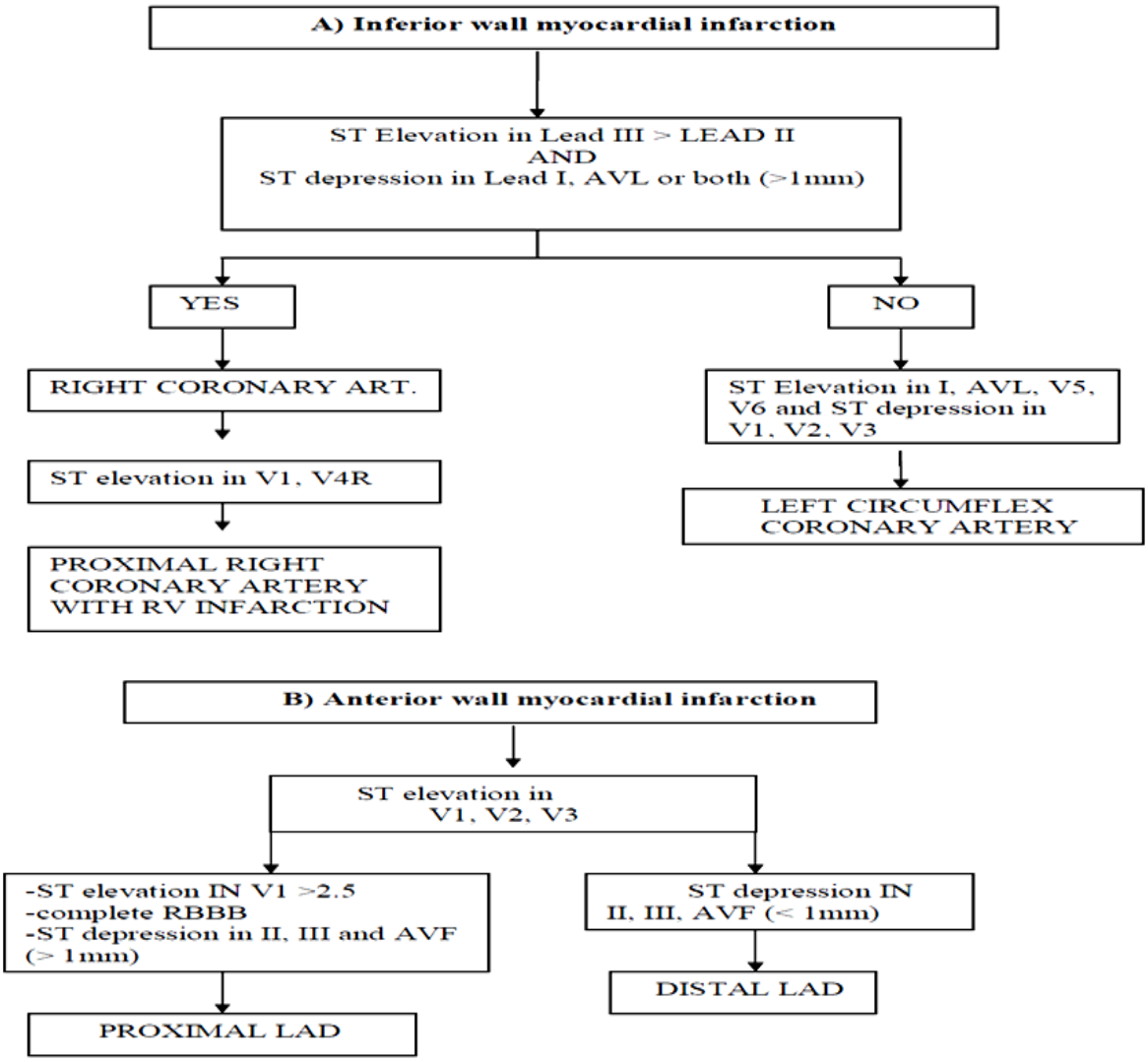
LABORATORY INVESTIGATIONS :

ELECTROCARDIOGRAM

The 12 lead electrocardiogram should be done for all patients presenting with chest pain as early as possible. It does not mean that a normal ECG does excludes the presence of myocardial infarction.The ECG provides critical information for both diagnosis and prognosis.

STEMI is characterized by at least 1 mm/ 1mv elevation of ST-segment in two or more contiguous chest leads. Reciprocal ST-segment depression in the leads away from the region of infarct further strengthens the diagnosis[9].

A right-sided ECG particularly RV3, RV4 should be obtained to exclude right ventricular infarction especially in inferior wall MI.



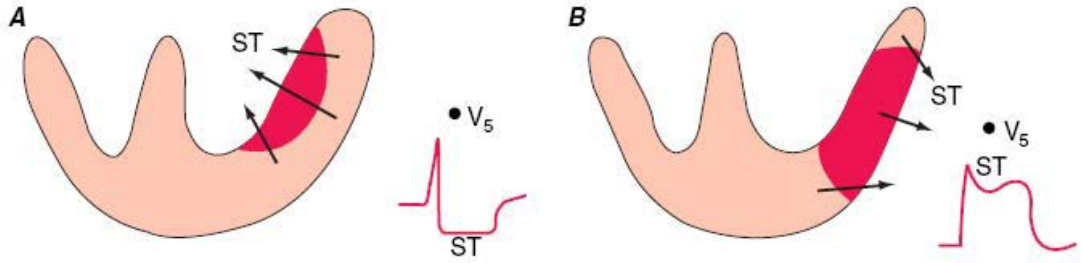
Marked ST depression in leads V_1 to V_3 with upright T waves indicates posterior wall infarction, which is confirmed by ST-segment elevation in VR7 to VR9 leads. This should be suspected in every case of inferior wall MI.

Patients presenting with new onset left bundle branch block fulfilling sgarbossa criteria can be managed as managed as anterior wall STEMI.

Other disease entities with associated ST-segment elevation should be kept in mind like pericarditis, myocarditis, left ventricular aneurysm, early repolarization, coronary artery

spasm, intracranial bleeding, head trauma and left ventricular apical ballooning syndrome.

Fig 6: SUBENDOCARDIAL AND TRANSMURAL INFARCT:



Patients with NSTEMI can present with marked or minimal ST-segment depression or isolated T wave changes. This identifies patients at higher risk who subsequently benefit most from an aggressive management strategy[10].

Nonspecific ST-segment and T wave abnormalities are usually defined as lesser amounts of ST-segment deviation or T wave inversion of 0.2 mV or less, and are less helpful for risk stratification .

Diffuse ST-segment elevation and PR-segment depression suggest pericarditis.

Fig 7: ST ELEVATION ANTERIOR AND INFERIOR WALL MI:

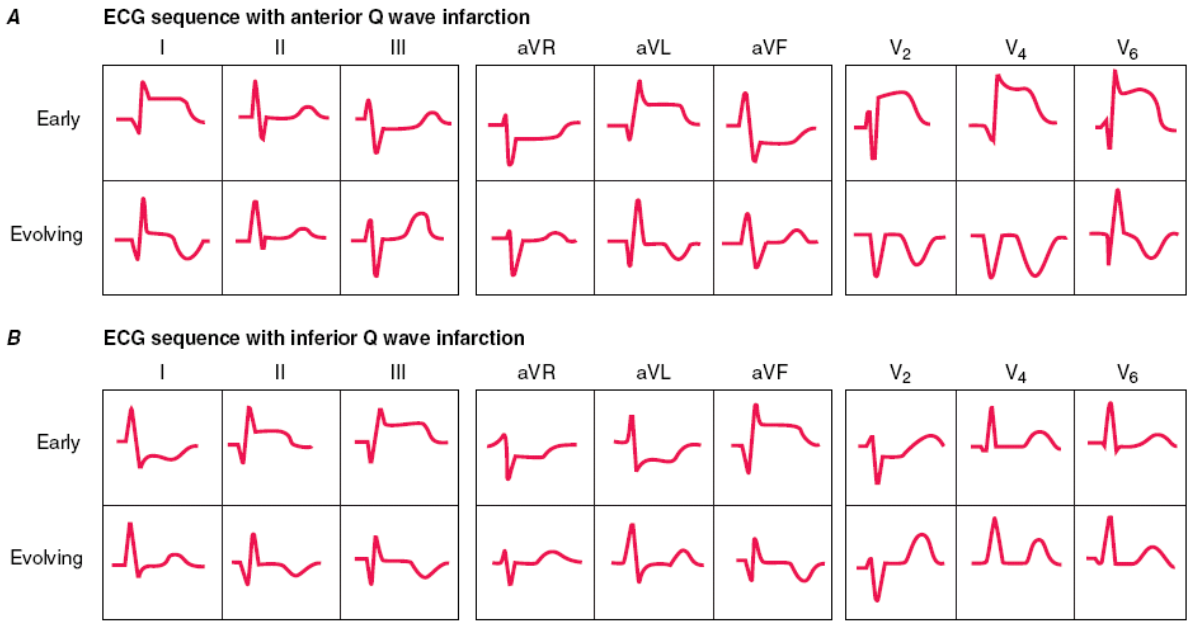


Table 1: Infarct-Related Artery and Distribution of ST-Segment Elevation[11]

Location of Coronary Occlusion	Distribution of ST-Segment Elevation	30-Day Mortality with Successful Reperfusion[*]
Proximal LAD (proximal to first septal perforator)	V_1-V_6, aVL, I, new LBBB common	19.6%
Middle LAD (distal to S1 branch and proximal to D1 branch)	V_1-V_6, aVL, I,	9.2%
Distal LAD (distal to origin of D1 branch) or diagonal branch	V_1-V_4, or aVL, I, V5-6	6.8%
Right coronary artery	II, III, aVF, V_{5-6} (V3R,	6.4%

	V4R with RV infarction)	
Left circumflex	V_{5-6}, III, II, aVF (can have minimal ECG changes)	4.5%

CHEST RADIOGRAPHY :

A chest radiograph is typically obtained in all patients presenting with chest pain. It is usually nondiagnostic of ACS but show pulmonary edema caused by ischemic diastolic or systolic dysfunction. It is more useful for diagnosing other disorders mimicking ACS for example; aortic dissection[12].

SERUM CARDIAC BIOMARKERS:

Cardiac bio markers are intracellular enzymes or proteins released into the blood in from necrotic dissolution of heart muscle after infarction[13].

Rapid kits for cardiac markers are now available widespread.

Table 2: FEATURES OF SERUM CARDIAC MARKERS ;

	Sensitivity at:					
Marker	**Time to Appearance**	**Duration of Elevation**	**6 hr**	**12 hr**	**Specificity**	**Comments**

Troponin I	2–6 hr	5–10 days	~75%	90–100%	~98%	Generally regarded as test of choice
Troponin T	2–6 hr	5–14 days	~80%	95–100%	~95%	Test of choice; less specific than troponin I (elevated in renal insufficiency)
CK-MB	3–6 hr	2–4 days	~65%	~95%	~95%	Test of choice for recurrent angina once troponin is elevated

(1) Creatine phosphokinase (CPK) :

It starts rising after four to eight 8 hours and falls to basal level by 48hours to 72 hours. It is more cheap and readily available. The only disadvantage is its non specific nature. It may be high in skeletal muscle disorders also. To overcome this issue is the use of ratio of MB subunit of creatine phosphikinase to the whole of creatine phosphokinase makes it more specific.

(2) Cardiac-specific troponin T (cTnT) and *troponin I* (cTnI)

These levels are undetectable in normal individuals but it is elevated in STEMI upto twenty times higher. They are now the preferred biochemical markers for a small MI that is below the detection limit for CKMB measurements. Their Levels remain high for 7 to 10 days

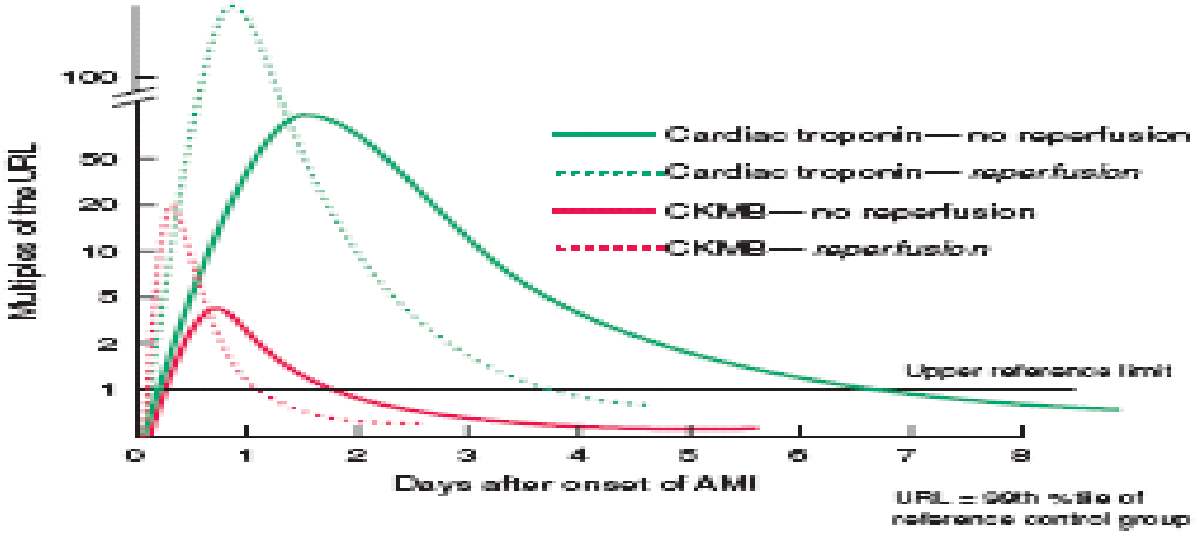
after the event. So they are less valuable in diagnosing reinfarction within one week.

(3) Myoglobin

It is the first enzyme to be released into the bllod after the onset of infarction. Because of its non specificity, rapid excretion, rapid fall of levels it is usually not preferred as a marker in myocardial infarction.

(4) Non specific markers like leukocytosis especially neutrophills and eosinophills which appears in the first day itself and persists for 3 to 7 days. The ESR rises more slowly ; peaks during the 1st week and remains elevated for 3 to 4 weeks.

FIG 8. PATTERN OF ENZYME ELEVATION IN MYOCARDIAL INFARCTION



CARDIAC IMAGING

ECHOCARDIGRAPHY

Random wall motion abnormalities in 2D ECHO are almost universally present.

The disadvantage is the acute STEMI cannot be differentiated from an old scar[14].

Echocardiography is useful in assessing left ventricular ejection fraction, diastolic dysfunction, mural thrombus, ventricular aneurysm, chamber hypertrophy.

It is also useful in diagnosing the complications like pericarditis, dressler syndrome, ventricular septal rupture and mgurgitation.

RADIONUCLIDE TECHNIQUE

(1) Myocardial perfusion imaging with 201Thallium or 99mTechnitium-sestamibi reveal a perfusion defect as cold spot in myocardial infaction. It can detect very earlier than enzymes but it is too expensive and it also cannot differentiate new oinfarction from old scar.

POSITRON EMISSION TOMOGRAPHY

PET scanning uses positron emitting agents to demonstrate alterations in the metabolism of myocardium. PET can easily differentiate stunned myocardium due to acute ischemia from an old scar .18 fluoro deoxy glucose is used as a marter . A nearby cyclotron is required to produce this 18 FDG tracer.

CORONARY ARTERIOGRAPHY

Coronary arteriography is indicated in (1) chronic stable angina people pectoris who are having severe symptoms inspite of adequate medical therapy(2) patients with troublesome symptoms and unconfirmed diagnosis in whom to rule out the diagnosis of IHD, (3) patients with angina pectoris who have experienced cardiac arrest and survived (4) patients with features of IHD on other non invasive testing with clinical or laboratory evidence of ventricular dysfunction[(16)] .

Noninvasive alternatives include CT angiography and cardiac MR angiography .Higher radiation exposure with CT angiography compared to conventional diagnostic arteriography and the artefacts on cardiac MR imposed by cardiac movement during the cardiac cycle especially at high heart rates are limitations.

STRESS TESTING:

An.All medications including beta blockers, nitrates should be discontinued one day prior to the test. The patient is allowed to exercise upto 85% of maximum predictable heart rate and a 12 lead ECG should be performed. A new ST depression of more than 2mm ; inability to exercise for more than two minutes; development of hypotension, arrhythmia, heart failure are the endpoints. stress testing can also be performed pharmacologically by using dipyridamole, adenosine and dobutamine [15].

RISK STRATIFICATION AND PROGNOSIS:

Table 3:Thrombolysis in MI Risk Score for Unstable Angina/NSTEMI

One Point score for each of the Following:	Score	Risk of Adverse Event[*]
Age ≥65 years	0/1	4.7%
Presence of =3 CV risk factors	2	8.3%
Recent (<24 h) severe angina	3	13.2%
Known coronary stenosis ≥50%	4	19.9%
ST-segment deviation on admission ECG ≥0.5 mm	5	26.2%

Use of aspirin within past 7 days	6/7	40.9%
Elevated biomarkers of cardiac injury (troponin, CK-MB)		

Abbreviations: CV = cardiovascular; ECG = electrocardiogram.

Table 4:Thrombolysis in MI Score for STEMI:

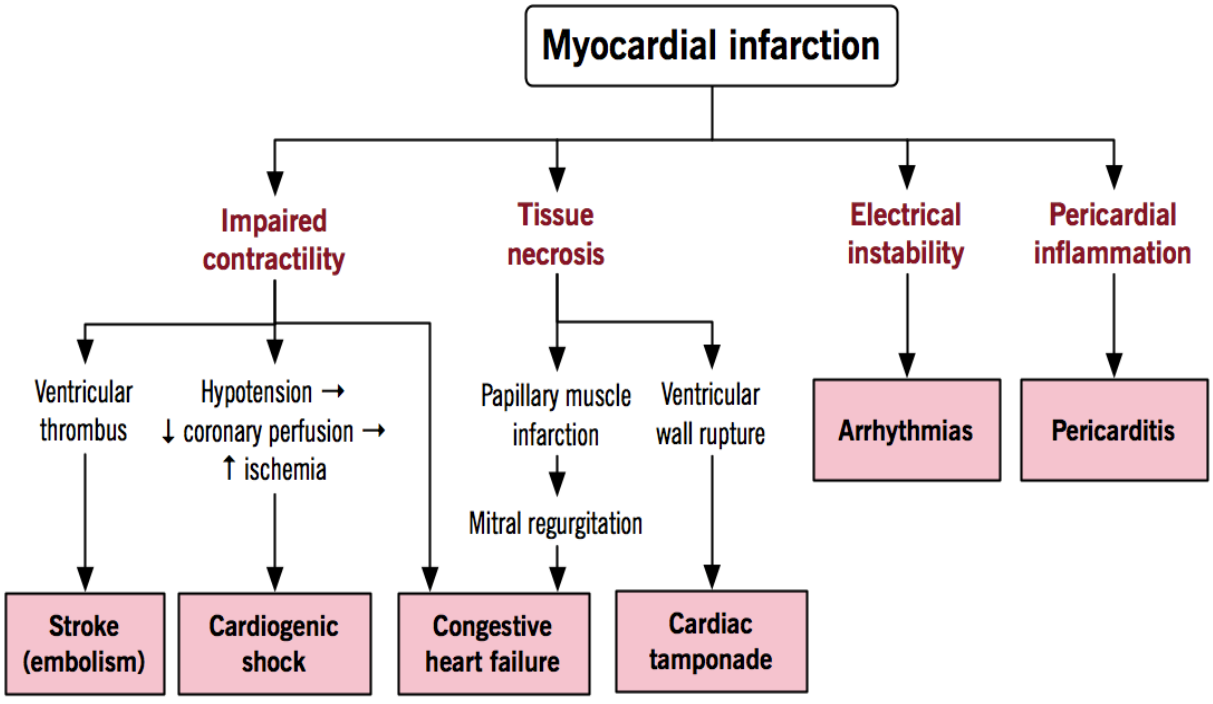
Various Risk Factors	**Points**	**Total Score**	**30-Day Mortality rate (%)**
Patient Age ≥75 years	3	0	0.8
Patient Age 65-74 years	2	1	1.6
Systolic Blood Pressure <100 mm Hg	3	2	2.2
Pulse rate>100 beats/min	2	3	4.4
Killip class more than 1	2	4	7.3
Anterior MI or LBBB	1	5	12.4

Diabetes mellitus, HTN, or angina	1	6	16.1
Weight <67 kg	1	7	23.4
Symptom onset to treatment >4 h	1	8	26.8
		>8	35.9

Abbreviations: BP = blood pressure; HTN =hypertension; LBBB = left bundle branch block; MI = myocardial infarction

COMPLICATIONS OF MYOCARDIAL INFARCTION:

Fig 9: VARIOUS COMPLICATIONS OF MYOCARDIAL INFARCTION:



TREATMENT:

The initial treatment of patients presenting with AMI are:

- Obtain intravenous (IV) access and stabilize hemodynamics if unstable.
- Relieve ischemic pain using IV morphine and sublingual or IV NTG.
- Minimize myocardial oxygen demand – supply mismatch with IV β-blockers (heart rate <70 bpm as blood pressure tolerates) and supplemental oxygen.
- Maintain myocardial perfusion using aspirin and heparin (either IV unfractionated or subcutaneous LMWH).

Patients with STEMI should receive prompt reperfusion therapy (thrombolysis or primary percutaneous intervention)

Aspirin

All patients should receive 324 mg of aspirin (four 81 mg tablets chewed and swallowed or as rectal suppository) and continue at 81 mg daily thereafter. In patients with STEMI, aspirin reduces mortality to a similar extent as thrombolytic therapy, Aspirin-allergic patients should receive 300 mg of clopidogrel.

Clopidogrel

Clopidogrel blocks the platelet ADP receptor .It is a prodrug converted into its active form in liver.A 300-mg loading dose is administered followed by 75 mg daily. Administer 600 mg loading of clopidogrel in ACS patients planned for Percutaneous coronary intervention. Clopidogrel combined with aspirin, compared with aspirin alone, reduces the risk of cardiovascular death, recurrent infarction, or stroke in patients with NSTEMI patients who

donot undergo percutaneous revascularization .Clopidogrel, 75 mg daily, should be continued for at least 9months -1year following percutaneous coronary revascularization. If coronary angiography is not anticipated, clopidogrel should be continued for 9 to 12 months.

Heparin

Patients presenting with Acute MI should be treated with either IV unfractionated heparin : 60 IU/kg intravenous bolus followed by 12 IU/kg /hour iv infusion or subcutaneous low-molecular-weight heparin (LMWH) except for STEMI patients receiving streptokinase. In STEMI, IVUFH is required to maintain vessel patency in those receiving a fibrin-specific thrombolytic agent (alteplase, reteplase, and tenecteplase). Administration ofLMWH reduces the risk of death and ischemic events compared with IV UFH in patients with NSTEMI and inunstable angina. LMWH appears safe when continued up until the time of coronary angiography and percutaneous intervention.LMWH should not be givento patients with significant renal failure (creatinine clearance < 50 mL / min) or morbid obesity[17].

β-Blockers

All patients with Acute MI should receive prompt oral β-blocker therapy to be continued indefinitely in the absence of contraindications. Metoprolol is preferred in the acute setting because of short half-life compared with atenolol. β-blockers are beneficial due to their ability to reduce oxygen supply-demand mismatch by reducing heart rate, reducing myocardial contraction, and reducing arterial BP . β-blockers also reduce the risk of atrial

and ventricular tachyarrythmias and reduce the risk of ventricular free wall rupture.

Nitroglycerin

Nitroglycerin can be administered as a sublingual formulation or as IV infusion. Nitroglycerin does not improve prognosis in AMI and should be used cautiously in patients with right ventricular or inferior wall MI that could result in hypotension. Nitroglycerin should not be given to the patients who have taken Viagra or other PDE inhibitor within 24 hours.

Glycoprotein IIb/IIIa Inhibitors

These agents block the GP IIb/IIIa platelet receptor, which functions as the receptor for fibrinogen adherence. They reduce ischemic complications associated with PCI and should be administered in patients for whom an early invasive strategy is planned. Benefit is shown with eptifibatide (Integrilin) and tirofiban (Aggrastat) in patients with NSTEMI ACS who do not undergo early PCI. This benefit appears isolated to high-risk patients including those with troponin elevation, ST-segment depression > 0.5 mV, diabetes mellitus, and LVEF< 40%.

HMG-CoA Reductase Inhibitors (Statins)

Early, aggressive statin therapy improves the clinical outcomes. Previous trials showed that intensive atorvastatin therapy with 80 mg daily initiated following ACS significantly reduces adverse cardiac events compared with less intensive statin therapy .A recent trial

(ARMYDA-ACS) demonstrated that pretreatment with atorvastatin 80 mg 12 hours prior to Percutaneous Coronary Intrvention gives around 90% reduction in adverse cardiac events and significantly reduced peri-procedural MI rates.

REPERFUSION THERAPY IN STEMI:

Regardless of the modality of reperfusion used, the time from symptom onset to myocardial reperfusion has the strongest influence on myocardial salvage, recovery of myocardial function, and improvements in mortality. Patients receiving successful reperfusion within three hours of symptom onset receives the greatest benefit.

Thrombolysis

Thrombolytic therapy is the most commonly utilized method of reperfusion worldwide. The fibrin-specific t-PA derived thrombolytic agents (alteplase reteplase and tenecteplase) have proved superior but significantly more expensive than the fibrin-nonspecific agents such as streptokinase. The fibrin-specific agents reduce 30-day mortality rate that is similar rates of successful reperfusion with streptokinase. Clinically,successful thrombolysis is associated with resolution of chest symptoms and reduction of ST-segment height by atleast50%. Patients with persistent symptoms, persistence of ST-segment elevation and hemodynamic collapse following thrombolysis should be referred for emergency PCI[(18).]

Thrombolytic Agents

- Streptokinase
- Reteplase

- Alteplase
- Tenecteplase

TABLE 5: CONTRAINDICATIONS OF THROMBOLYSIS[19]:

Contraindications for Thrombolytic Administration
Absolute
• Prior ICH
• Ischemic stroke less than 3 months duration
• Ongoing active bleeding anywhere except menstruation
• Significant head or facial injury less than 3 months
• Possible or doubtful aortic dissection
• Intracranial neoplasm
• Intracranial vascular structural abnormality
Relative
• Internal bleed in the recent past i.e within 4 week
• Major internal surgery within 3 week
• History of ischemic stroke

- prolonged Cardio Pulmonary Resuscitation of more than 10 minutes
- Pregnancy
- Patients on anticoagulants with INR >2.0
- Noncompressible vascular puncture
- Accelerated hypertension BP >180/110 mm Hg
- Active peptic ulcer disease
- Prior streptokinase exposure (for repeat streptokinase administration)

PERCUTANEOUS CORONARY INTERVENTION (PCI) IN STEMI:

Primary PCI including coronary artery stenting of the artery is superior to thrombolysis when done by experienced operators in high-volume centers with less ("door-to-balloon") time i.e < 90 min.

Primary PCI is the treatment of choice in patients with contraindications to thrombolytic therapy. Stenting; along with the glycoprotein IIb/IIIa antagonist abciximab—is now widely used in patients with acute myocardial infarction.

In the patients with cardiogenic shock, early catheterization and percutaneous or surgical revascularization are the preferred mode of approach now a days. This carries a lower risk of hemorrhagic complications.

Facilitated PCI is an approach in which a combination of fibrinolytic agents either in full or half the dose is given and is followed by immediate PCI[(20).]

METABOLIC ABNORMALITIES IN DIABETES:

ABNORMAL MUSCLE AND FAT METABOLISM

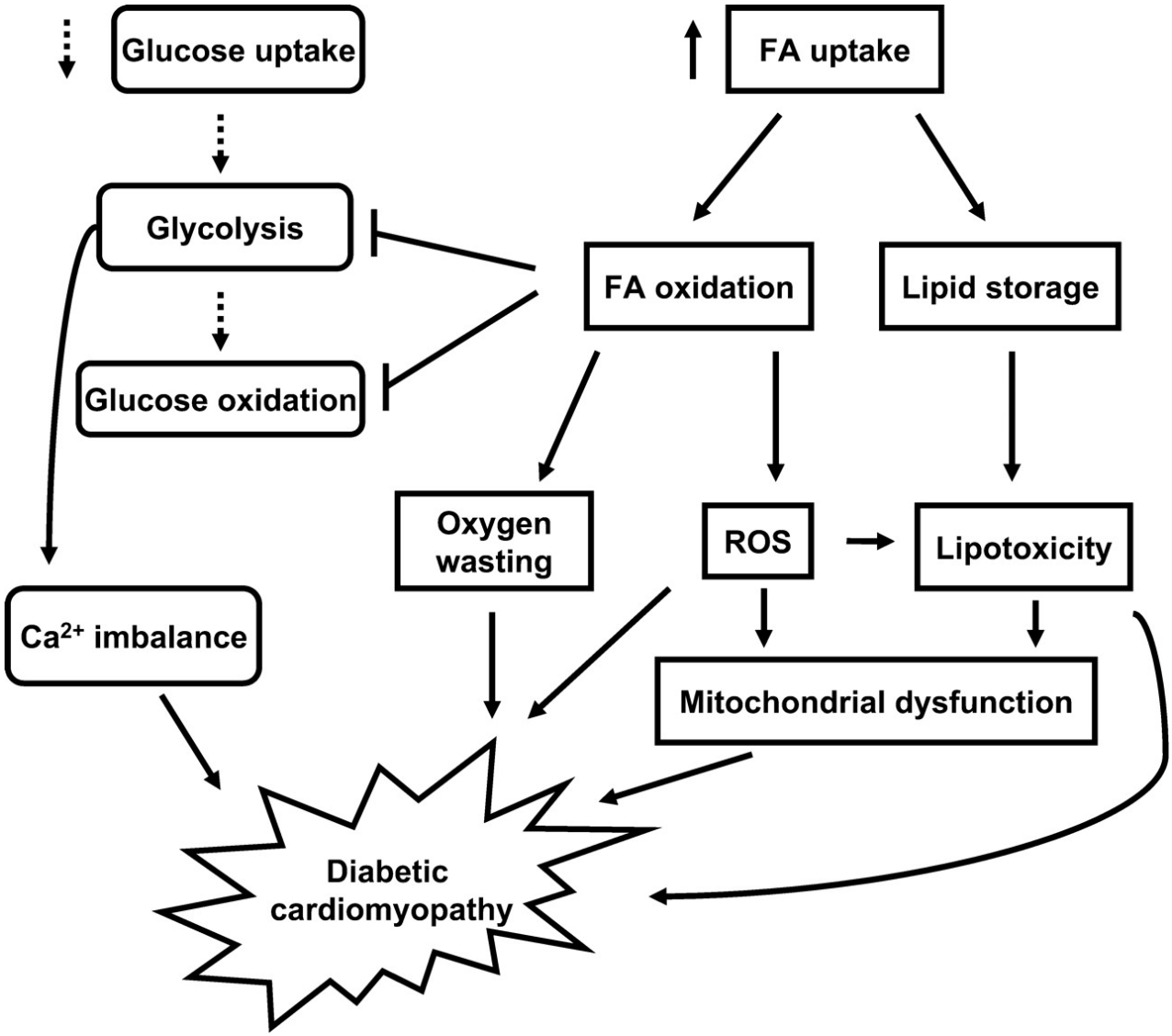
Insulin resistance, the decreased ability of insulin to act effectively on target tissues (especially muscle, liver, and fat), is a prominent feature of type 2 DM and results from a combination of genetic susceptibility and obesity. Insulin resistance is relative, however, since supranormal levels of circulating insulin will normalize the plasma glucose. Insulin dose-response curves exhibit a rightward shift, indicating reduced sensitivity, and a reduced maximal response, indicating an overall decrease in maximum glucose utilization (30–60% lower than in normal individuals). Insulin resistance impairs glucose utilization by insulin-sensitive tissues and increases hepatic glucose output; both effects contribute to the hyperglycemia. Increased hepatic glucose output predominantly accounts for increased FPG levels, whereas decreased peripheral glucose usage results in postprandial hyperglycemia. In skeletal muscle, there is a greater impairment in nonoxidative glucose usage (glycogen formation) than in oxidative glucose metabolism through glycolysis. Glucose metabolism in insulin-independent tissues is not altered in type 2 DM[(21).]

The precise molecular mechanism leading to insulin resistance in type 2 DM has not been elucidated. Insulin receptor levels and tyrosine kinase activity in skeletal muscle are reduced, but these alterations are most likely secondary to hyperinsulinemia and are not a primary defect. Therefore, "postreceptor" defects in insulin-regulated phosphorylation/dephosphorylation appear to play the predominant role in insulin

resistance. For example, a PI-3-kinase signaling defect might reduce translocation of GLUT4 to the plasma membrane. Other abnormalities include the accumulation of lipid within skeletal myocytes, which may impair mitochondrial oxidative phosphorylation and reduce insulin-stimulated mitochondrial ATP production. Impaired fatty acid oxidation and lipid accumulation within skeletal myocytes also may generate reactive oxygen species such as lipid peroxides. Of note, not all insulin signal transduction pathways are resistant to the effects of insulin (e.g., those controlling cell growth and differentiation using the mitogenic-activated protein kinase pathway). Consequently, hyperinsulinemia may increase the insulin action through these pathways, potentially accelerating diabetes-related conditions such as atherosclerosis.[22]

The obesity accompanying type 2 DM, particularly in a central or visceral location, is thought to be part of the pathogenic process. The increased adipocyte mass leads to increased levels of circulating free fatty acids and other fat cell products .For example, adipocytes secrete a number of biologic products (nonesterified free fatty acids, retinol-binding protein 4, leptin, TNF- , resistin, and adiponectin). In addition to regulating body weight, appetite, and energy expenditure, adipokines also modulate insulin sensitivity. The increased production of free fatty acids and some adipokines may cause insulin resistance in skeletal muscle and liver. For example, free fatty acids impair glucose utilization in skeletal muscle, promote glucose production by the liver, and impair beta cell function. In contrast, the production by adipocytes of adiponectin, an insulin-sensitizing peptide, is reduced in obesity, and this may contribute to hepatic insulin resistance. Adipocyte products and

adipokines also produce an inflammatory state and may explain why markers of inflammation such as IL-6 and C-reactive protein are often elevated in type 2 DM. In addition, inflammatory cells have been found infiltrating adipose tissue. Inhibition of inflammatory signaling pathways such as the nuclear factor B (NF-kB) pathway appears to reduce insulin resistance and improve hyper-glycemia in animal models.[23]



IMPAIRED INSULIN SECRETION:

In type 2 DM, insulin secretion initially increases in response to insulin resistance to maintain normal glucose tolerance. Initially, the insulin secretory defect is mild and

selectively involves glucose-stimulated insulin secretion. The response to other nonglucosesecretagogues, such as arginine, is preserved. Abnormalities in proinsulin processing is reflected by increased secretion of proinsulin in type 2 diabetes. Eventually, the insulin secretory defect progresses to a state of inadequate insulin secretion.

The reason(s) for the decline in insulin secretory capacity in type 2 DM is unclear. The assumption is that a second genetic defect—superimposed upon insulin resistance—leads to beta cell failure. Beta cell mass is decreased by approximately 50% in individuals with long-standing type 2 diabetes. Islet amyloid polypeptide or amylin is co-secreted by the beta cell and forms the amyloid fibrillar deposit found in the islets of individuals with long-standing type 2 DM. Whether such islet amyloid deposits are a primary or secondary event is not known. The metabolic environment of diabetes may also negatively impact islet function. For example, chronic hyperglycemia paradoxically impairs islet function ("glucose toxicity") and leads to a worsening of hyperglycemia. Improvement in glycemic control is often associated with improved islet function. In addition, elevation of free fatty acid levels ("lipotoxicity") and dietary fat may also worsen islet function.

INCREASED HEPATIC GLUCOSE AND LIPID PRODUCTION

In type 2 DM, insulin resistance in the liver reflects the failure of hyperinsulinemia to suppress gluconeogenesis, which results in fasting hyperglycemia and decreased glycogen storage by the liver in the postprandial state. Increased hepatic glucose production occurs early in the course of diabetes, though likely after the onset of insulin secretory

abnormalities and insulin resistance in skeletal muscle. As a result of insulin resistance in adipose tissue, lipolysis and free fatty acid flux from adipocytes are increased, leading to increased lipid [very low density lipoprotein (VLDL) and triglyceride] synthesis in hepatocytes[44]. This lipid storage or steatosis in the liver may lead to nonalcoholic fatty liver disease and abnormal liver function tests. This is also responsible for the dyslipidemia found in type 2 DM [elevated triglycerides, reduced high-density lipoprotein (HDL), and increased small dense low-density lipoprotein (LDL) particles].

INSULIN RESISTANCE SYNDROMES

The insulin resistance condition comprises a spectrum of disorders, with hyperglycemia representing one of the most readily diagnosed features. The *metabolic syndrome*, the *insulin resistance syndrome*, or *syndrome X* are terms used to describe a constellation of metabolic derangements that includes insulin resistance, hypertension, dyslipidemia (decreased HDL and elevated triglycerides), central or visceral obesity, type 2 diabetes or IGT/IFG, and accelerated cardiovascular disease[41].

A number of relatively rare forms of severe insulin resistance include features of type 2 DM or IGT (Table 344-1). Mutations in the insulin receptor that interfere with binding or signal transduction are a rare cause of insulin resistance. Acanthosisnigricans and signs of hyperandrogenism (hirsutism, acne, and oligomenorrhea in women) are also common physical features. Two distinct syndromes of severe insulin resistance have been described in adults: (1) type A, which affects young women and is characterized by severe

hyperinsulinemia, obesity, and features of hyperandrogenism; and (2) type B, which affects middle-aged women and is characterized by severe hyperinsulinemia, features of hyperandrogenism, and autoimmune disorders. Individuals with the type A insulin resistance syndrome have an undefined defect in the insulin-signaling pathway; individuals with the type B insulin resistance syndrome have autoantibodies directed at the insulin receptor. These receptor autoantibodies may block insulin binding or may stimulate the insulin receptor, leading to intermittent hypoglycemia.

Polycystic ovary syndrome (PCOS) is a common disorder that affects premenopausal women and is characterized by chronic anovulation and hyperandrogenism . Insulin resistance is seen in a significant subset of women with PCOS, and the disorder substantially increases the risk for type 2 DM, independent of the effects of obesity.

HYPERGLYCEMIA IN MYOCARDIAL INFARCTION:

The mortality and morbidity of the diabetic patient sustaining a myocardial infarction is poor compared with that of non-diabetic patients.Studies have shown that hyperglycemia induces adverse prognosis even in non diabetic patients . Claure Bennard observed and explained acute hyperglycemic response to stress more than a century ago. “Diabetes of injury” concept evolved as glucose has been identified as metabolic mirror of the severity and outcome of critical illness.

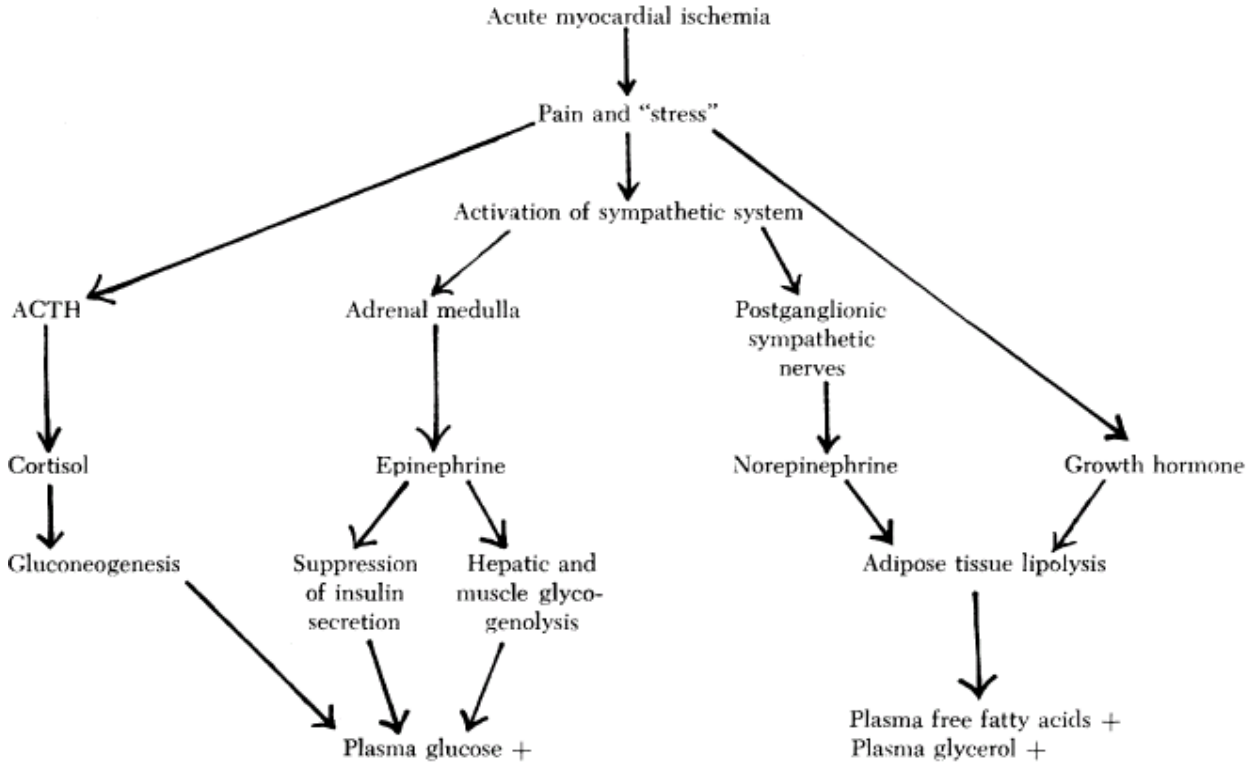
PREDICTIVE VALUE OF PLASMA GLUCOSE IN MYOCARDIAL INFARCTION[24]:

- Elevated admission glucose levels in patients with acute myocardial infarction is independently associated with large infarct sizes.
- A significant assosiation between hyperglycaemia at the time of infarction and the development of heart failure has been reported in large scale studies.
- Admission Hyperglycemia in MI is found to be associated with high heart rate in Acute Myocardial infarction.

MECHANISM OF HYPERGLYCEMIA IN MI:

The exact pathological mechanism of hyperglycaemia induced by Acute MI is unclear, although several explanations have been proposed

FIG 10: MECHANISM OF STRESS HYPERGLYCEMIA:



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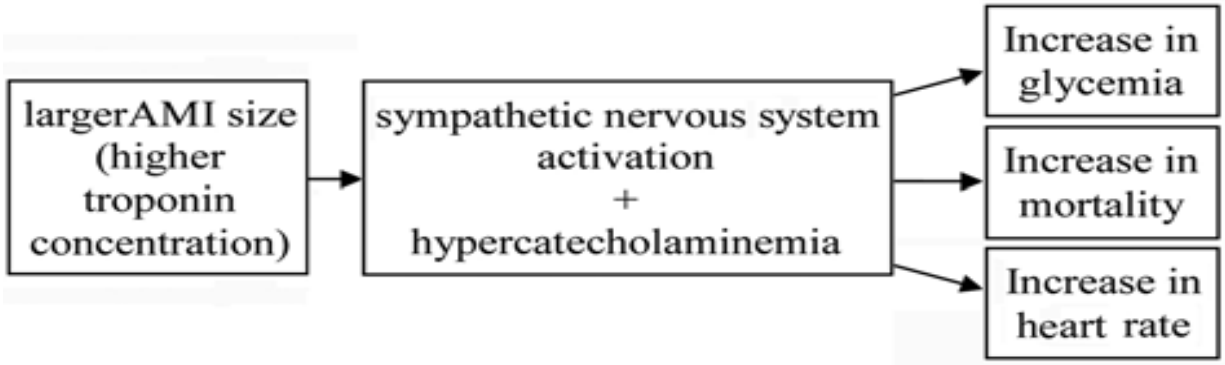
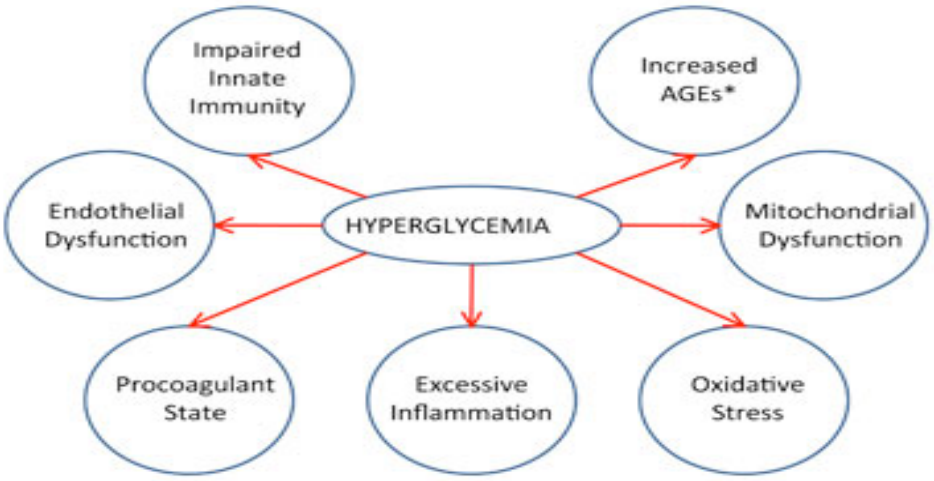
Stress during the occurrence of myocardial infarction induces the secretion of adrenal steroids that have various adverse effects like increasing heart rate ; increasing myocardial oxygen demand; increasing glycogenolysis in the liver; suppressing insulin production; augmenting glucagon secretion.The resulting elevated blood sugar has adverse consequences on myocardium[25].



VARIOUS EFFECTS OF ACUTE HYPERGLYCEMIA ON Acute MI:

Fig 11: VARIOUS ADVERSE EFFECTS OF HYPERGLYCEMIA IN MI



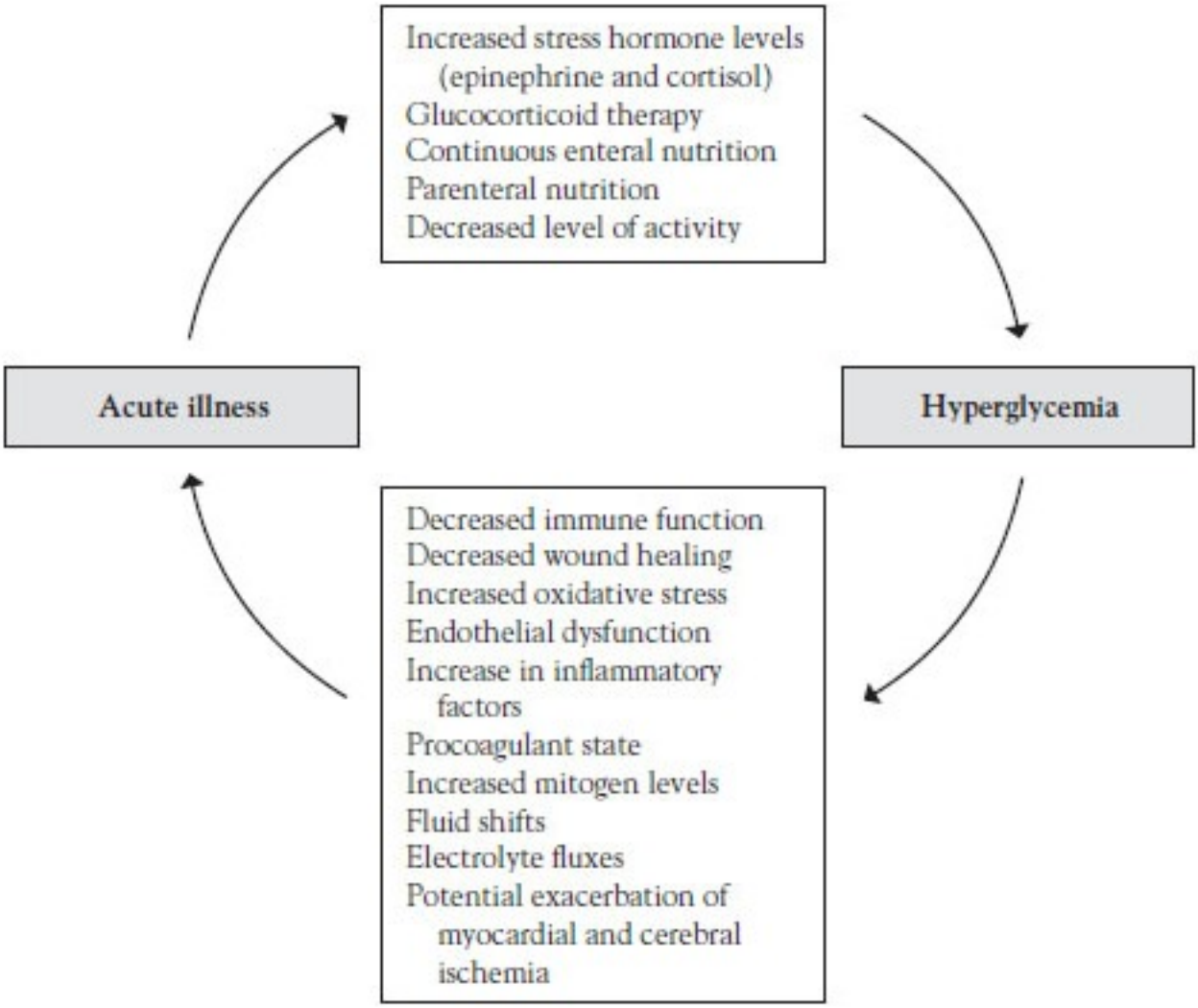
1. Electrophysiological changes:

High blood gucose content in the blood induces some changes in resting membrane potential of cardiac myocytes. These changes produces a state of electrical instability which is superimposed on altered conduction during myocardial infarction. The final result will be occurrence of arrhythmias which could be fatal sometimes e.g Ventricular tachycardia.

2. Impaired left ventricular function:

Acute hyperglycemia causes impaired left ventricular systolic function which has been proved in various studies. The probable mechanism is the induction of vasospasm by glucose thet prevents the blood flow during reperfusion. Further hyperglycemia shifts the metabolism from glycolysis to fatty acid metabolism.

FIG 12: MECHANISM OF HYPERGLYCEMIC MYOCARDIAL INJURY:



3. Effect on coagulation:

IHyperglycemia by unknown mechanism induces platelet activation and aggregation; activation of extrinsic factors of coagulation and inhibits fibrinolysis.The net result is the prothrombotic state that promotes coronary occlusion.

4. Effect on inflammatory immune reaction:

Atherosclerosis itself is considered as a state of systemic inflammation. These patients are in a state of ongoing inflammation that rapidly progress to a peak level during the event of acute coronary syndrome. Acute MI patients usually have elevated levels of C-Reactive protein, ESR, Interleukin-18, Tumour Necrosis Factor in their blood[(27).] These substances

further causes damage of cardiac myocytes.

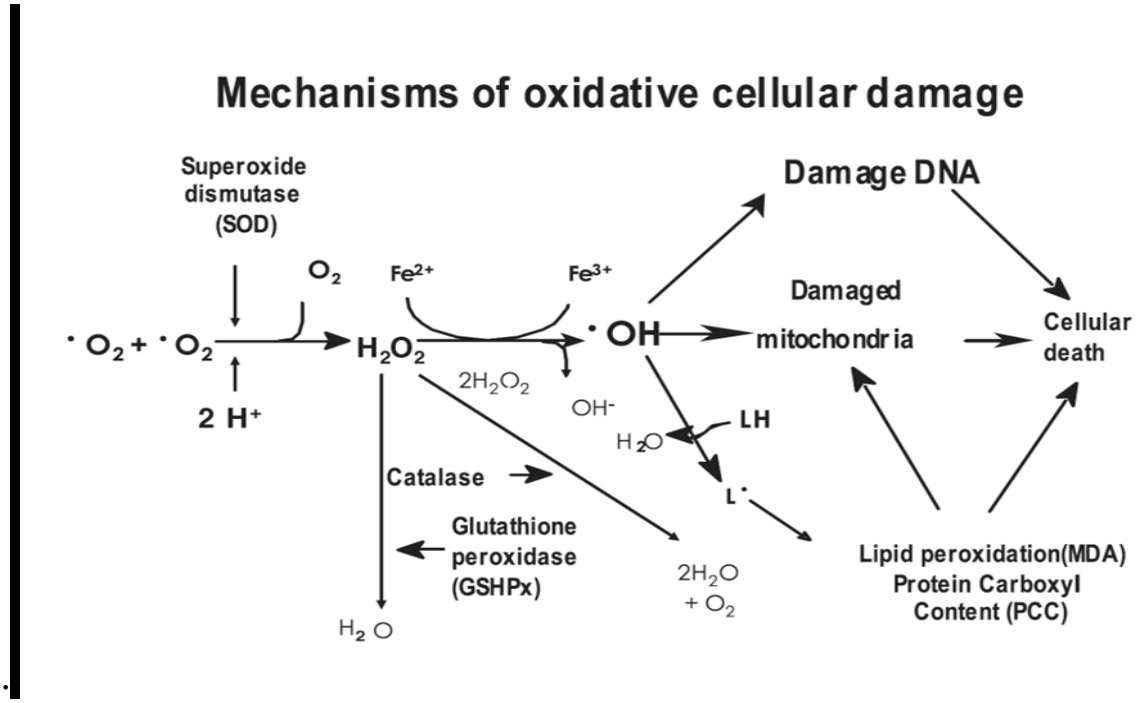
5. Effect on endothelial function:

Hyperglycemia diminishes the ability of endothelium to secrete nitric oxide which is a potent vasodilator. Further damage to the vascular endothelium favours platelet activation and aggregation leading to the expansion of thrombus[26].

6. Oxidative stress:

Oxidative stress is a well known pathogenic process for atherosclerosis and cardiovascular diseases. Free radical production by hyperglycemia is the probable pathologic process. Free radical injury is more common during the stage of reperfusion. It cause depletion of intracellular glutathione stores, mitochondrial dysfunction, lipid peroxidation of membranes and DNA damage. 3-Nitrotyrosine , a marker of oxidative damage and it has recently been demonstrated to be increased in monkeys during acute hyperglycemia[29]

Fig 13: MECHANISM OF HYPERGLYCEMIC OXIDATIVE DAMAGE:



LEUKOCYTES:

Leukocytes are the nucleated blood cells which are mainly involved in the immune functions of the body[30]. They are synthesized in the bone marrow (the granulocytes , monocytes) and in the lymphoid tissue (lymphocytes and plasma cells).

TYPES OF LEUKOCYTES

They are :

1) Polymorphonuclear neutrophils

2) Polymorphonuclear eosinophils

3) Polymorphonuclear basophils

4) Monocytes

5) Lymphocytes

6) Plasma cells

CAUSES OF LEUKOCYTOSIS[31]

NEUTROPHILIC LEUKOCYTOSIS

1) Acute bacterial infections

2) Tissue necrosis like burns and myocardial infarction.

EOSINOPHILIC LEUKOCYTOSIS

1) Parasitic infestations

2) Malignancies – Hodgkins and NHL

3) Vasculitis

4)Allergic disorders

BASOPHILIC LEUKOCYTOSIS

Myeloproliferative disorders - CML

MONOCYTOSIS

1) Chronic infections – tuberculosis

2) Bacterial endocarditis

3) Rickettsiosis

4) Malaria

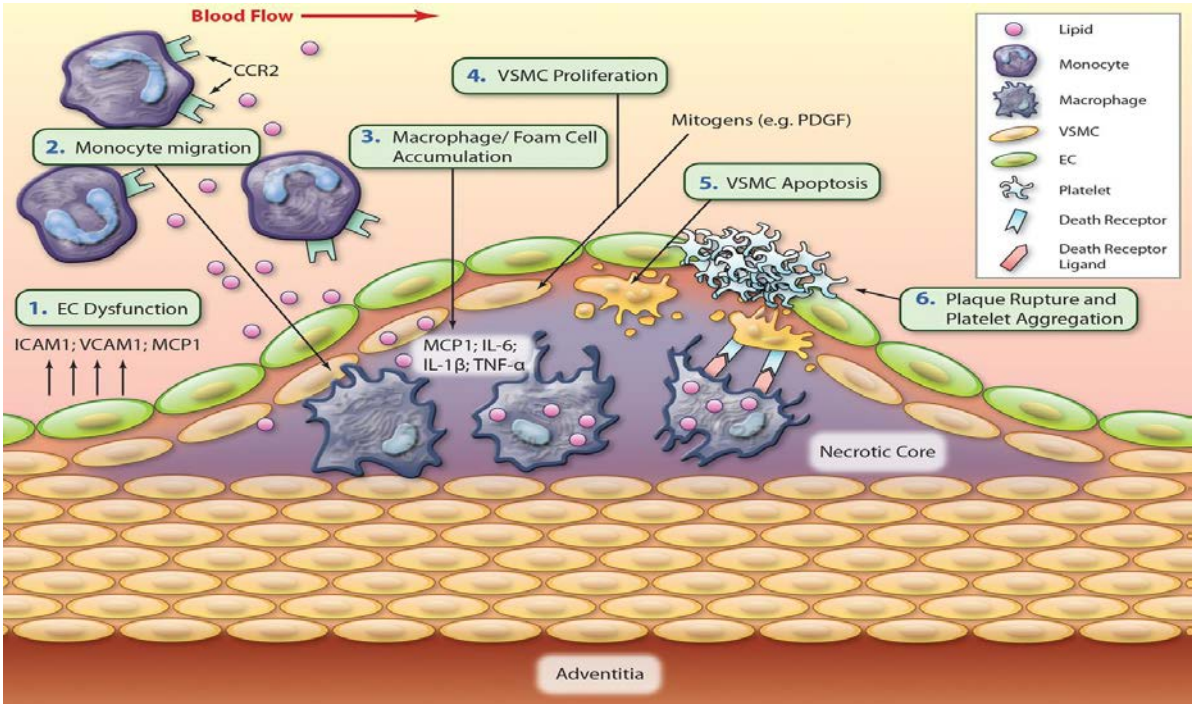
5) Collagen vascular diseases (S.L.E)

6) Inflammatory bowel disorders

ROLE OF LEUKOCYTES IN ATHEROSCLEROSIS[32]:

After endothelial injury by an unknown initiating factor, white blood cells especially monocytes undergo margination and attach themselves to the vascular endothelium. These cells migrate into the sub endothelial space by diapedesis and gets deposited. This initiates the recruitment of more and more leukocytes into that area. These cells secrete some enzymes that converts LDL into oxidized LDL. These particles are taken up by the residing macrophages and gets converted into foam cells. The cytokines like TGF beta released from these cells causes the proliferation of smooth muscle cells in the tunica media and mediates their migration into fibrous cap of atheromatous plaque. The lipid core will enlarge in course of time and finally the Matrix Metallo Proteases , collagenase, elastase causes the degradation of the fibros cap that exposes the subendothelial core . The platelets gets attached and activated that leads to thrombus formation and vessel lumen obstruction.

Fig 13: ROLE OF WBC IN ATHEROSCLEROSIS:



MECHANISM OF MYOCADIAL INJURY BY LEUKOCYTES[33]:

1)Endothelial injury

2)Luminal obstruction

3)Reduced myocyte blood supply

4)Exaggerated leukocyte recruitment

5)Increased expression of monocyte chemoattractant protein

6)Clotting system overactivity

7)Electrical instability

8)Increased leukocyte adhesion.

Proteolytic and oxidative vascular damage

This is mainly mediated by polymorphs. These cells secrete elastase, hyaluronidase,

collagenase and other proteolytic enzymes that causes detachment of vascular endothelial cells. This makes the platelets easy for their attachment in the exposed subendothelium and formation of thrombus. chemokines released from these cells recruites more leukocytes. Free radicals released from these cells further causes endothelial damage.

Vessel plugging

The intensification of inflammation causes synthesis and recruitement of more lymphocytes that are abnormal in their function and morphology. These cells are more rigid , large in size, more sticky that makes them to plug and occlude the blood vessel. The narrowing of lumen by the plaque further worsens the situation[(36).]

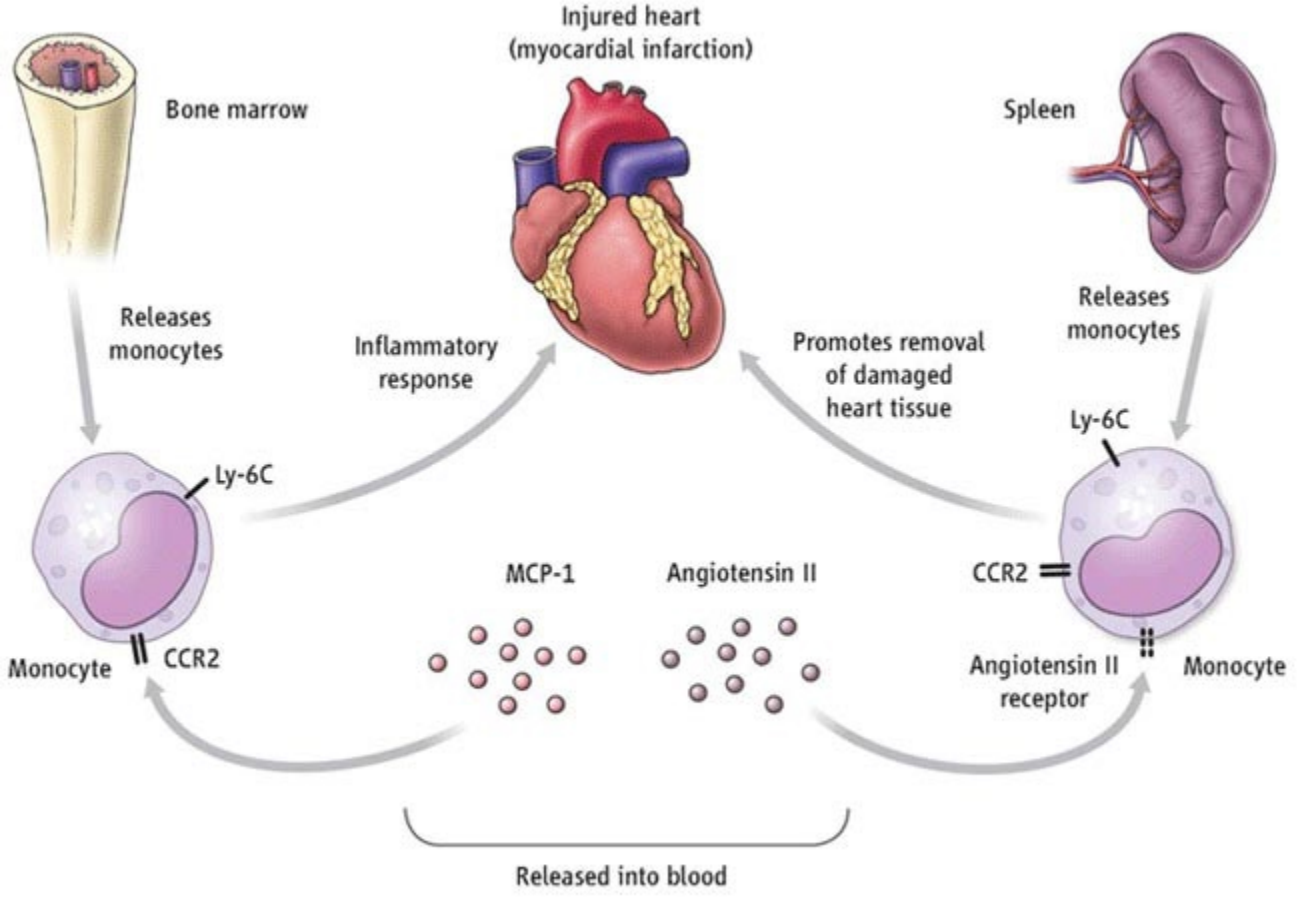
Abnormal leukocyte aggregation

Leukocytes also have surface receptors similar to other cells. These receptors get activated in the presence of chemokines tht leads to their stasis , aggregation , and destruction that leads to further release of mediators.Rise in hematocrit also contributes to the effect.

Leukocytes and infarct expansion

The mechanism of infarct expansion includes two different phenomenon. After the blood flow is restored more leukocytes enter into the affected area that aggravates the microvascular plugging and prevents the reflow pattern. The other one is the entry of more leukocytes and increased oxygen tension in that area leads to production of more reactive oxygen species that further damages cardiac myocytes and endothelium through free radical mediated injury.These processes contributes to the expansion of infarction.

Fig 14: ROLE OF INFLAMMATORY CYTOKINES IN MYOCARDIAL INJURY:



Leukocytes and hypercoagulability[34]

There is a linear association between the total leukocyte count and the coagulant activity of the blood. The leukocytes especially monocytes are involved in the activation of platelets. These monocytes can also take part in forming the platform for coagulation cascade. Leukocytes activates the extrinsic clotting factors especially factor 7 and factor 8. Interleukins released from these cells also activates complement system that promotes coagulation.

Leukocytes and reperfusion

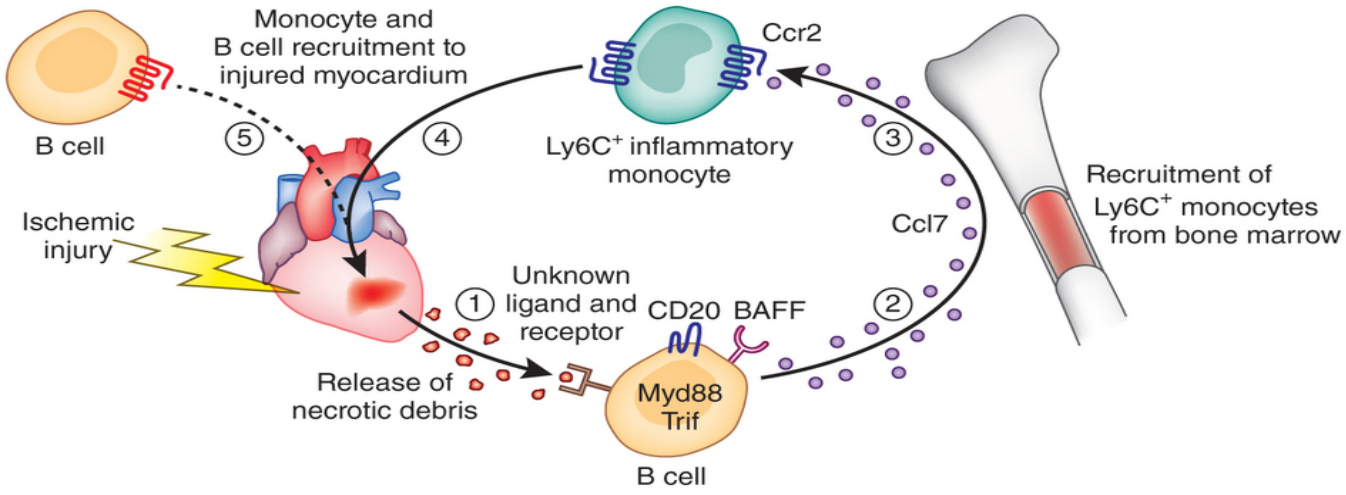
High leukocyte count increases the thrombin formation through the interaction between P

selectin , vascular endothelial adhesion molecule, and intercellular adhesion molecule. This increased thrombin burden resist the physiological fibrinolytic system.

Leukocytes and electrical instability

Leukocyte through the free radicals alter the membrane potential of cardiac myocytes and the superimposed altered pattern conduction that results in VT and VF.

Fig :15 MECHANISM OF LEUKOCYTOSIS IN MYOCARDIAL INFARCTION:



Myocardial infarction results in the release of inflammatory cytokines from the necrotic tissue that get attached to the activated B cell through CD 20 and some unknown ligand . This process releases lymphokines that recruits more number of lymphocytes and monocytes from the bonemarrow resulting in myocardial injury

Free Radical Injury:

ROS are capable of causing irreversible damage to all biochemical compounds Of the cell including lipids, nucleic acids, proteins, carbohydrates and connective tissues. ROS has an effect on membrane function also. ROS are thought to be involved in

causation of many clinical conditions like acute glomerulonephritis, cancer, atherosclerosis and Parkinson's disease.

Oxidative stress is capable of inducing oxidation of lipids and in the presence of oxygen, lipid peroxidation of cell membranes. It is generally accepted that lipid oxidation proceeds via a free radical induced mechanism called autooxidation, which induces initiation, propagation, and termination stages and predominantly occurs with PUFA. Polyunsaturated acyl chains of membrane phospholipids are particularly sensitive to lipid peroxidation. Lipid oxidation, both nonenzymatic and enzymatic, is self propagating in cellular membrane. Peroxidation of PUFA is classically depicted as a series of three or four basic reactions; however, the process becomes more complex as both the degree of unsaturation and severity of peroxidative conditions increases[35].

Lipid peroxidation causes loss of membrane fluidity, membrane potential, increases the membrane permeability of Ca2+ and causes loss of membrane integrity. MDA is formed by fatty acids with three or more double bonds and is used as a measure of lipid peroxidation

MATERIALS AND METHODS

SOURCE OF DATA

80 consecutive patients presenting with acute ST Elevation myocardial infarction admitted to IMCU/ ICCU Thanjvur Medical College Hospital from January 2014 to August 2014 were studied.

SAMPLE SIZE

Size of the sample n= 80

p = prevalence rate

q = 1-p

INCLUSION CRITERIA

Patients admitted in IMCU/ICCU

1)presenting within 48 hrs of symptom onset.

2) Anginal Chest pain lasting more than 30 minutes.

3) Characteristic ECG changes with ST segment elevation more than 1mm in limb leads or more than 2mm in two contiguous chest leads

EXCLUSION CRITERIA

1**)**Patients presenting after 48 hours of symptom onset.

2)Patients receiving drugs/iv fluids elevating blood glucose levels.

3)Post surgical or post traumatic up to one month.

COLLECTION OF DATA:

Random blood glucose level was measured at the time of admission. It is estimated by

glucose oxidase glucose peroxidase technique.

3ml of EDTA mixed blood sample was collected and subjected to complete blood count – both total and differential count by automated coulter counter and by manual examination.

PATIENT STRATIFICATION:

Patients were grouped into 3 categories according to their admission blood glucose levels,

Blood glucose Group I: Random blood sugar < 130 mg%,

Blood glucose Group II: Random blood sugar 131- 180 mg%, and

Blood glucose Group III: Random blood sugar >I80 mg%

Patients were grouped into 3 categories according to their admission blood glucose levels,

WBC Group I: Total count < 8000 cells/ cu mm.

WBC Group II: Total count 8000-11000/ cu mm and

WBC Group III: Total count >11000 cu/mm.

STUDY END POINTS :

The primary end point of the study is all cause mortality during the period of stay in the hospital.

STATISTICAL METHODS:

Data was analyzed by using following statistical methods,

1. Diagrammatic (bar / pie chart)representation

2. Mean + standard deviation (SD)

3. Chi square test for non continuous variables

4. Analysis of variance for continuous variables

5. Multivariate analysis tests to determine the association between W.B.C count and blood glucose levels with in hospital mortality.

INVESTIGATIONS

A) Blood

Haemoglobin

Total count

Differential count

B) Urine:

Albumin

Sugar

Microscopy

C) Biochemistry

Random blood glucose on admission

Urea ,

Serum creatinine

D) 12 lead Electrocardiography (ECG)

E) 2D Echocardiography – Ejection fraction

RESULTS AND STATISTICS:

Table 6: AGE DISTRIBUTION OF PATIENTS:

Particulars	No.of respondents (n=80)	Percentage (100%)
Below 40yrs	7	8.8
41 to 50yrs	14	17.5
51 to 60yrs	29	36.3
61 to 70yrs	21	26.3
71yrs & above	9	11.3

Graph1: BAR DIAGRAM OF AGE DISTRIBUTION OF PATIENTS :

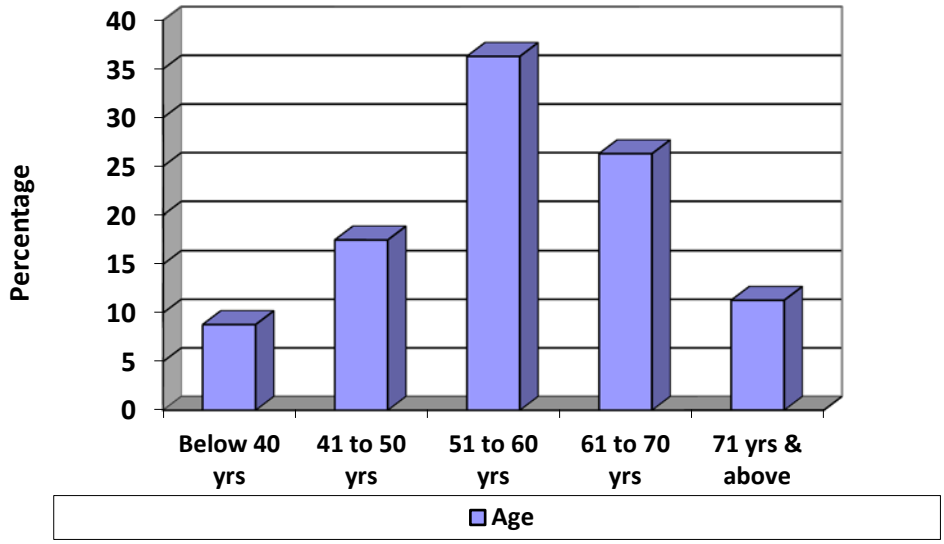


Table 7: SEX DISTRIBUTION OF PATIENTS:

Particulars	No.of respondents (n=80)	Percentage (100%)
Male	62	77.5
Female	18	22.5

Graph2: BAR DIAGRAM OF SEX DISTRIBUTION OF PATIENTS

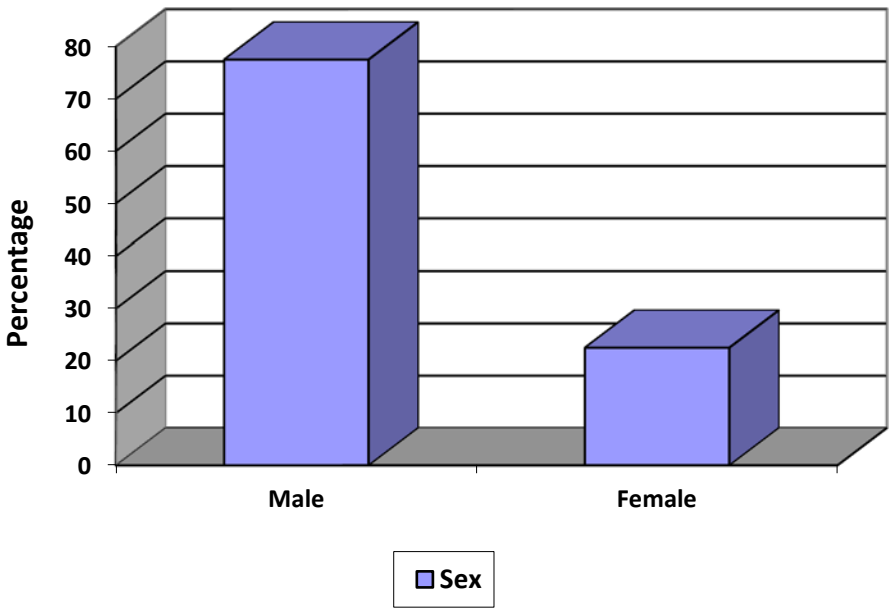


Table 8: DISTRIBUTION OF SMOKERS IN STUDY GROUP:

Particulars	No.of respondents (n=80)	Percentage (100%)
No	33	41.3
Yes	47	58.8

Graph3: DISTRIBUTION OF SMOKERS IN STUDY GROUP:

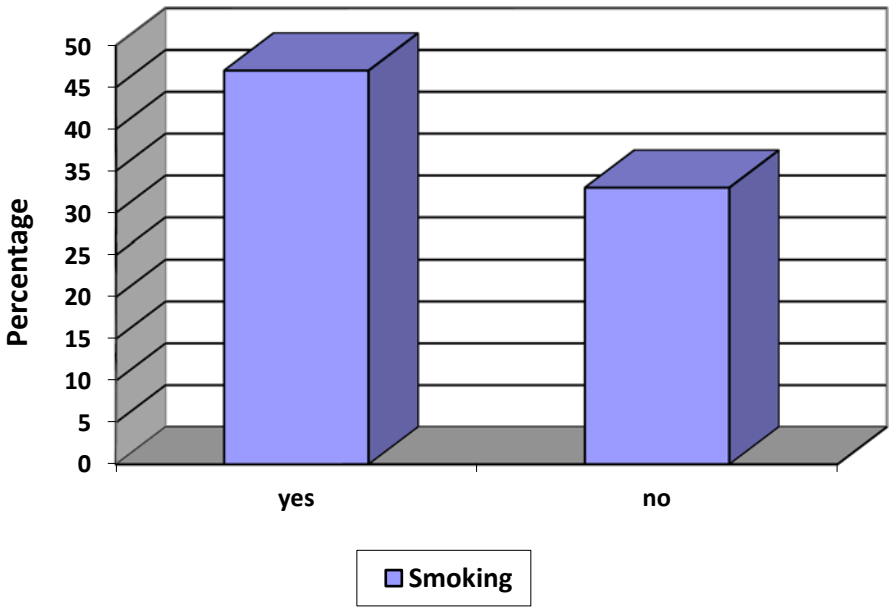


Table 9: DISTRIBUTION OF DIABETICS IN STUDY GROUP:

Particulars	No.of respondents (n=80)	Percentage (100%)
No	31	38.8
Yes	49	61.3

Graph4: DISTRIBUTION OF DIABETICS IN STUDY GROUP:

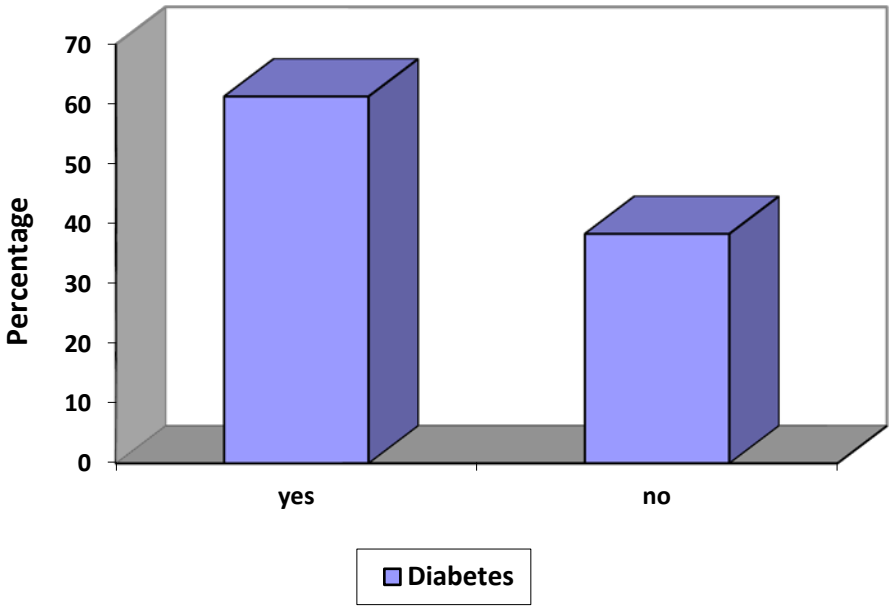


Table 10: DISTRIBUTION OF HYPERTENSIVE PATIENTS IN STUDY GROUP:

Particulars	No.of respondents (n=80)	Percentage (100%)
No	37	46.3
Yes	43	53.8

Graph5: DISTRIBUTION OF HYPERTENSIVE PATIENTS IN STUDY GROUP:

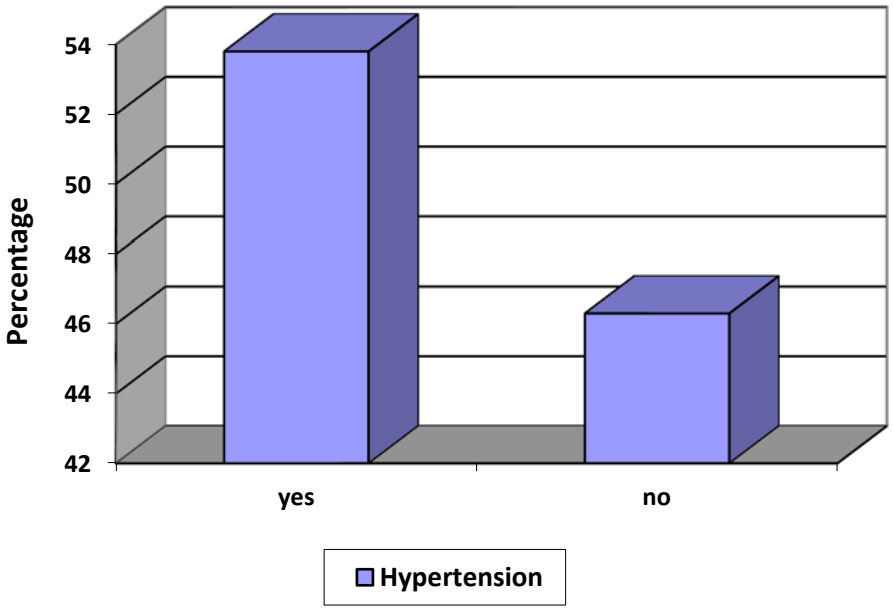


Table 11: DISTRIBUTION OF KNOWN IHD PATIENTS IN STUDY GROUP:

Particulars	No.of respondents (n=80)	Percentage (100%)
No	36	45.0
Yes	44	55.0

Graph6: DISTRIBUTION OF KNOWN IHD PATIENTS IN STUDY GROUP:

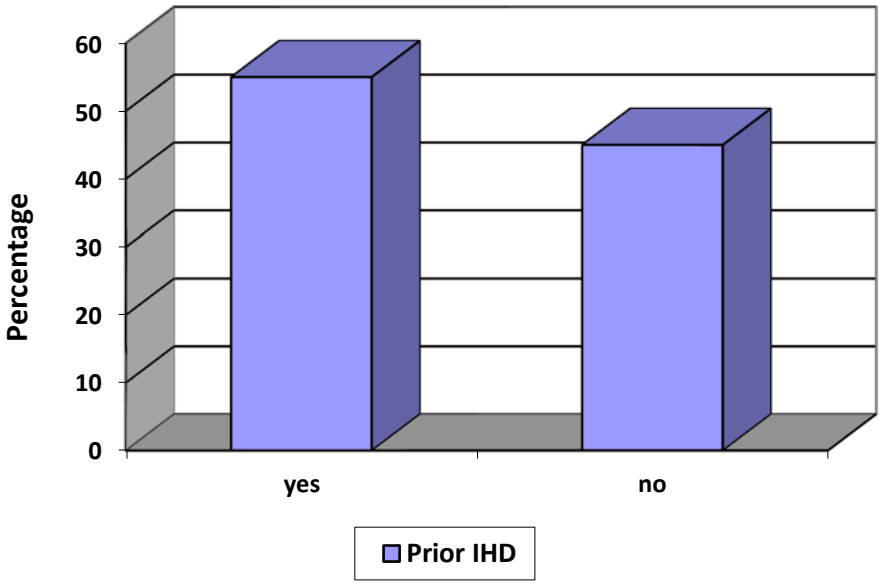


Table 12: DISTRIBUTION OF PATIENTS ACCORDING TO KILLIP CLASS:

Particulars	Number (n=80)	Percentage (100%)
1	31	38.8
2	31	38.8
3	14	17.5
4	4	5.0

Graph7: DISTRIBUTION OF PATIENTS ACCORDING TO KILLIP s

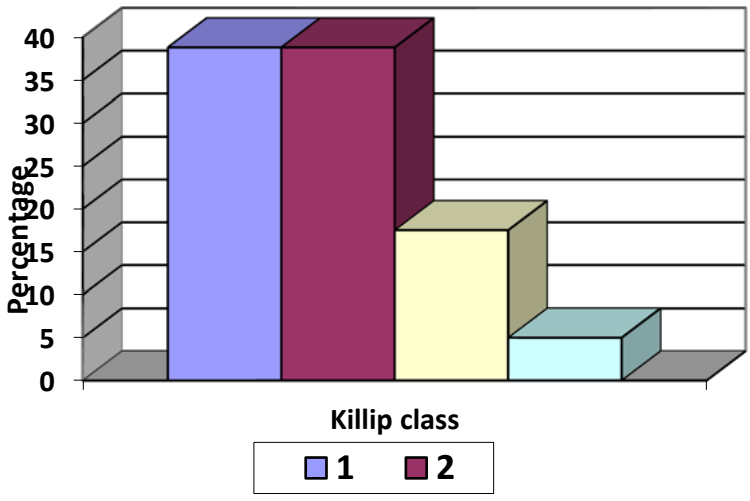


TABLE 13: DISTRIBUTION OF THE THROMBOLYSED PATIENTS:

Particulars	No.of respondents (n=80)	Percentage (100%)
Negative	26	32.5
Positive	54	67.5

Graph8: DISTRIBUTION OF THE THROMBOLYSED PATIENTS:

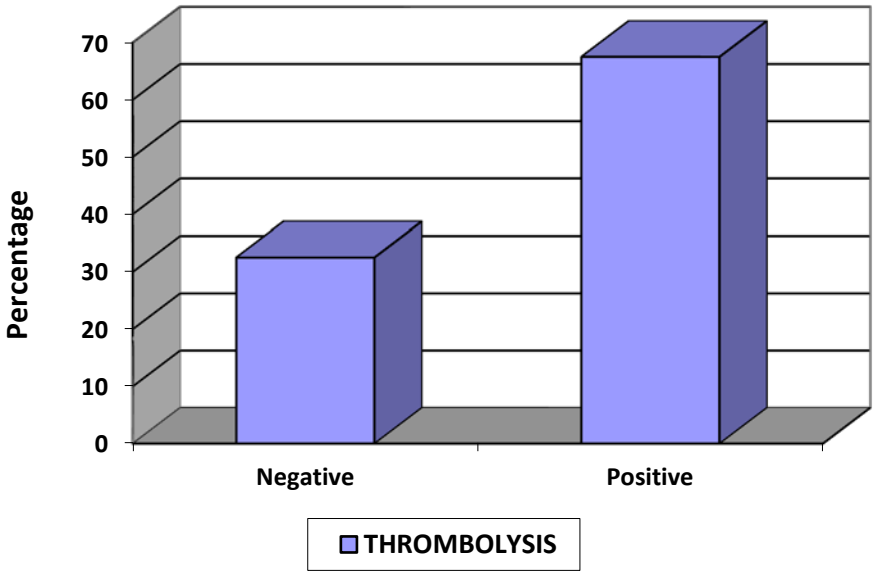


Table 14: DISTRIBUTION OF PATIENTS IN WBC <8000 GROUP

WBC< 8000 & (RBS<130)

Particulars	**No.of subjects (n=80)**	**Percentage (100%)**
No	65	81.3
Yes	15	18.8

WBC< 8000 & (RBS130-180)

Particulars	**No.of respondents (n=80)**	**Percentage (100%)**
No	66	82.5
Yes	14	17.5

In the WBC group1 i.e. WBC count <8000/ cu mm. the number of deaths in RBS<130 mg subgroup was 0 ; in RBS 130- 180mg subgroup was 1(6.7%) and in RBS>180mg subgroup was 4(26.7%) and the chi square analysis showed there is a significant association of the last subgroup with the mortality (p = 0.017).

WBC< 8000 & (RBS>180)

Particulars	No.of respondents (n=80)	Percentage (100%)
No	72	90.0
Yes	8	10.0

Graph9: DISTRIBUTION OF PATIENTS IN WBC <8000 GROUP

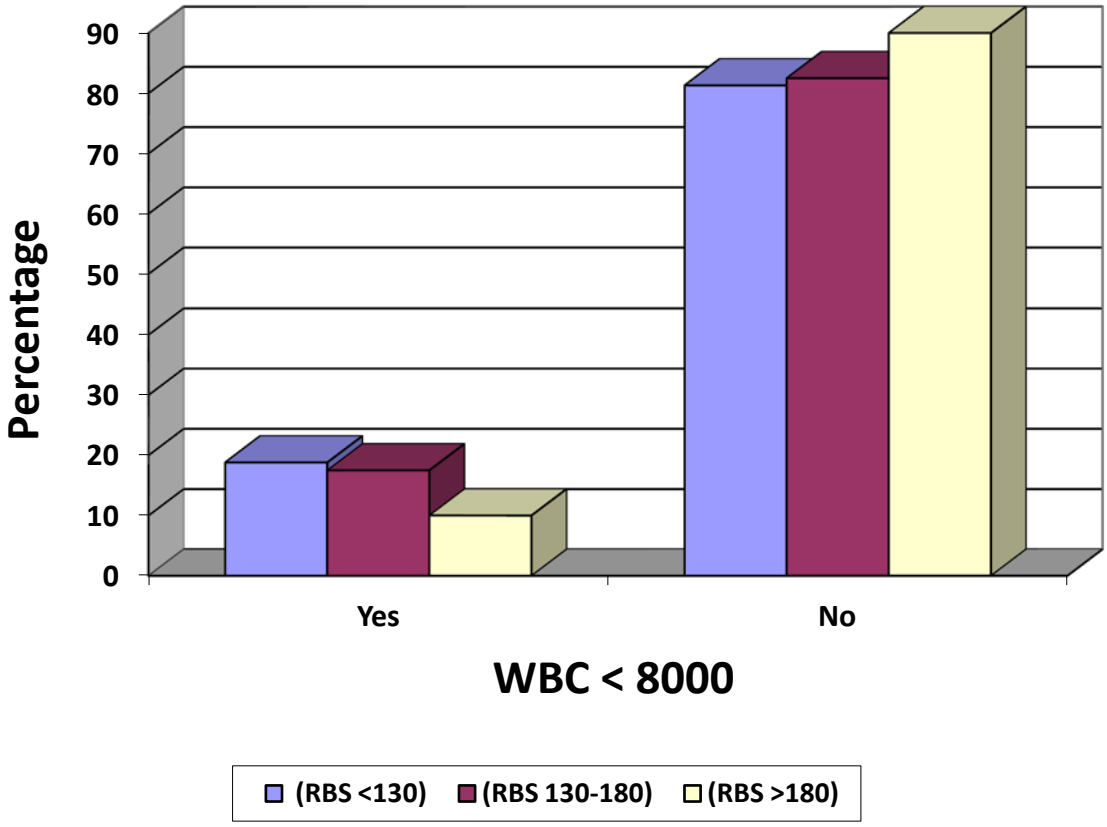


Table 15: DISTRIBUTION OF PATIENTS IN WBC 8000- 11000 GROUP

WBC- 8000-11000 (RBS<130)

Particulars	No.of respondents (n=80)	Percentage (100%)
No	64	80.0
Yes	16	20.0

WBC- 8000-11000 (RBS130-180)

Particulars	No.of respondents (n=80)	Percentage (100%)
No	68	85.0
Yes	12	15.0

In the WBC group2 i.e. WBC count 8000-11000 cells / cu mm. the number of deaths in RBS<130 mg subgroup was 0 ; in RBS 130- 180mg subgroup was 3(20.0%) and in RBS>180mg subgroup was 4(26.7%) and the chi square analysis showed there is a significant association of the 2^{nd} and 3^{rd} subgroup with the mortality (p = 0.039) and (p = 0.032) respectively.

WBC- 8000-11000 (RBS>180)

Particulars	No.of respondents (n=80)	Percentage (100%)
No	75	93.8
Yes	5	6.3

Graph 10::DISTRIBUTION OF PATIENTS IN WBC 8000- 11000 GROUP

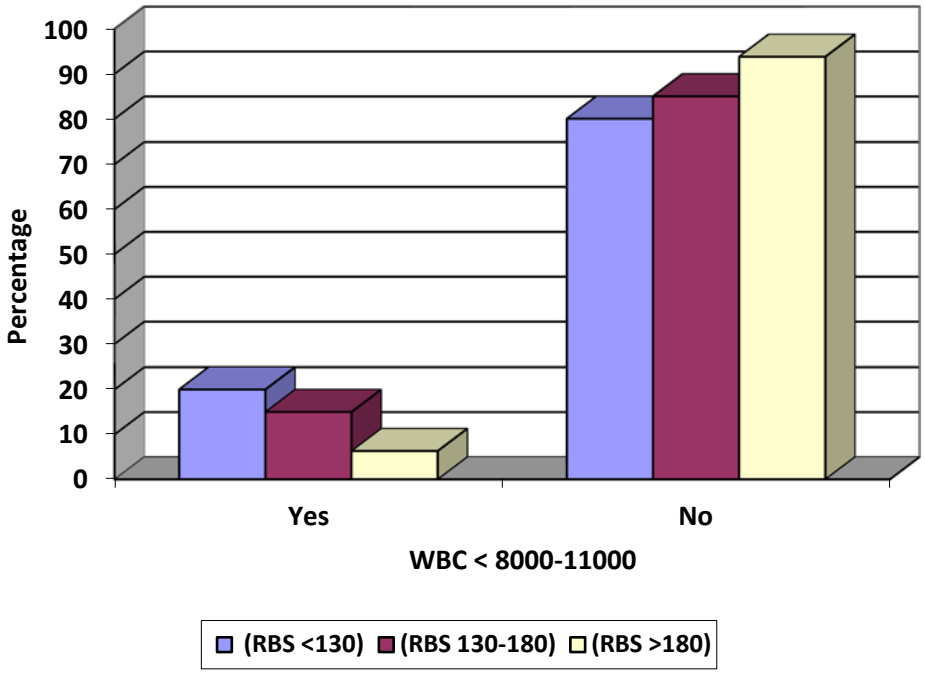


Table 16: DISTRIBUTION OF PATIENTS IN WBC >11000 GROUP

WBC> 11000 (RBS<130)

Particulars	No.of respondents (n=80)	Percentage (100%)
No	78	97.5
Yes	2	2.5

WBC> 11000 (RBS130-180)

Particulars	No.of respondents (n=80)	Percentage (100%)
No	75	93.8
Yes	5	6.3

In the WBC group3 i.e. WBC count >11000/ cu mm. the number of deaths in RBS<130 mg subgroup was 0 ; in RBS 130- 180mg subgroup was 1(6.7%) and in RBS>180mg subgroup was 7(46.7%) and the chi square analysis showed there is a significant association of the last subgroup with the mortality (p = 0.001)

WBC> 11000 (RBS>180)

Particulars	No.of respondents (n=80)	Percentage (100%)
No	73	91.3
Yes	7	8.8

Graph 10: DISTRIBUTION OF PATIENTS IN WBC >11000 GROUP

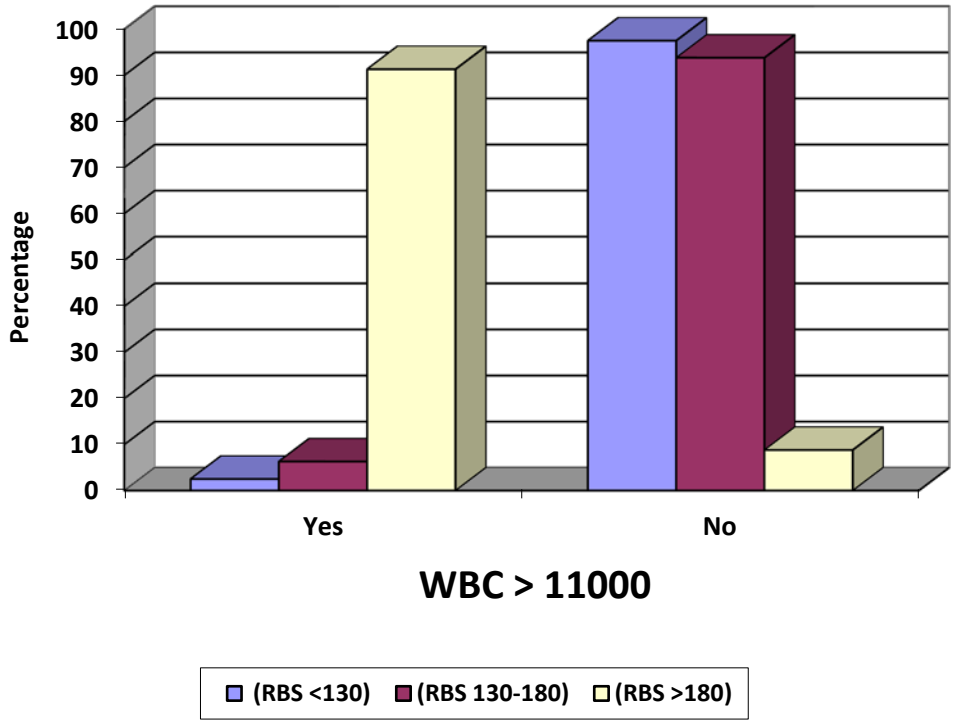


Table 17: FREQUENCY TABLE OF TERRITOTY INVOLVED:

Particulars	No.of respondents (n=80)	Percentage (100%)
ASMI	3	3.8
AWMI	50	62.5
IWMI	25	31.3
RVMI	2	2.5

Graph 11:FREQUENCY OF TERRITOTY INVOLVED:

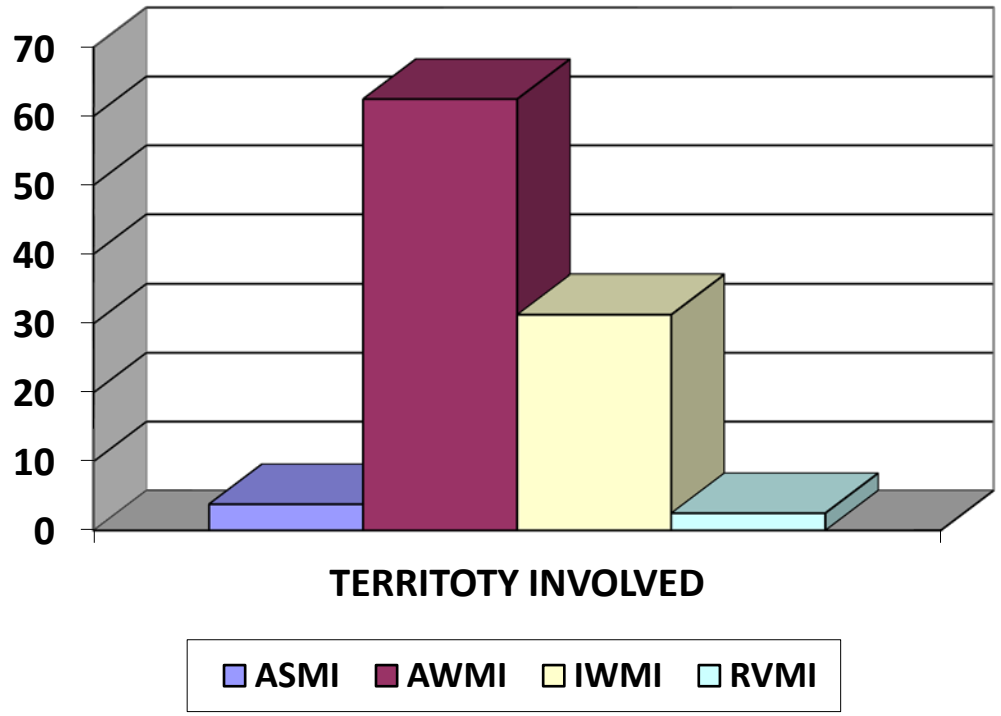
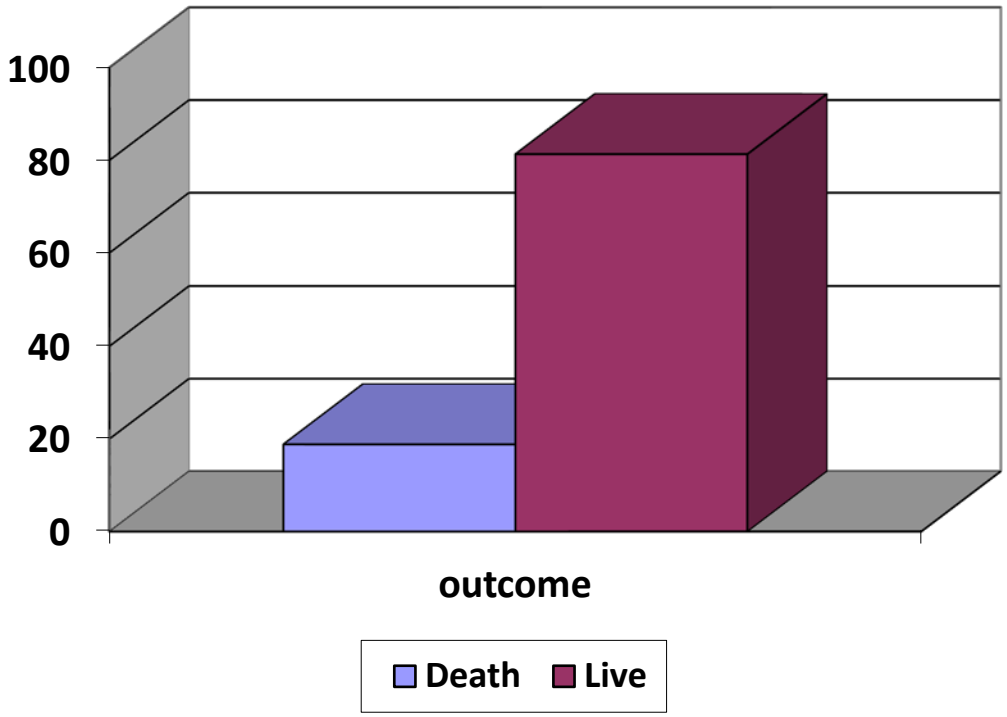


TABLE 18: OUTCOME -DEATH(D) RECOVERY (R)

Particulars	No.of respondents (n=80)	Percentage (100%)
Death	15	18.8
Live	65	81.3

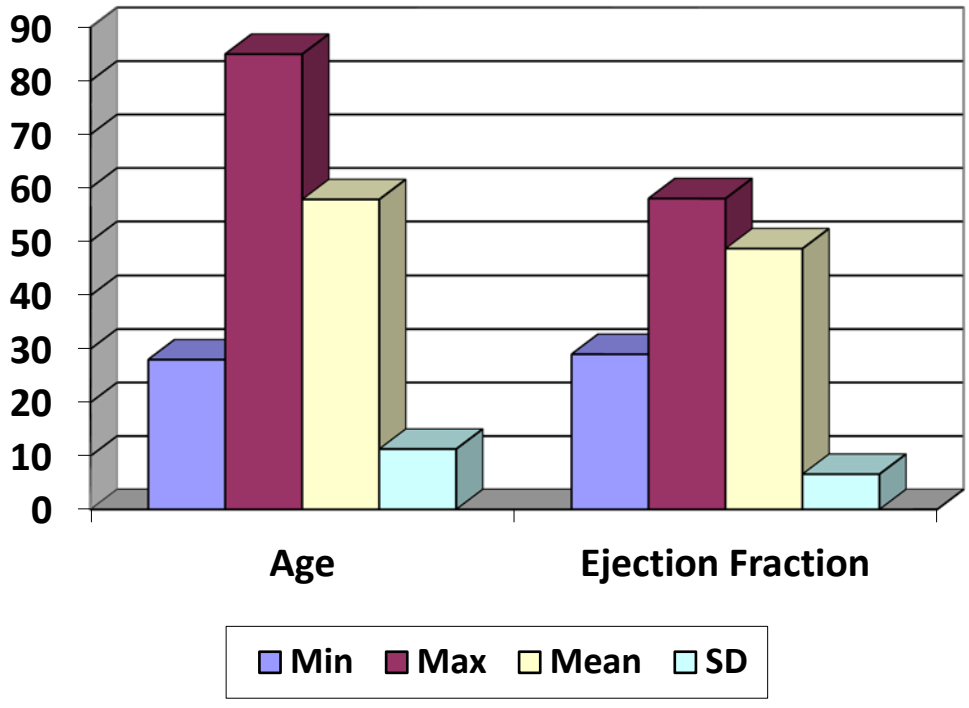
Graph 12: BAR DIAGERM SHOWING THE OUTCOME:



Tab19: FREQUENCY DISTRIBUTION OF AGE & EJECTION FRACTION:

Item	Min	Max	Mean	S.D
AGE	28	85	57.83	11.280
EJECTION FRACTION(%)	29	58	48.64	6.592

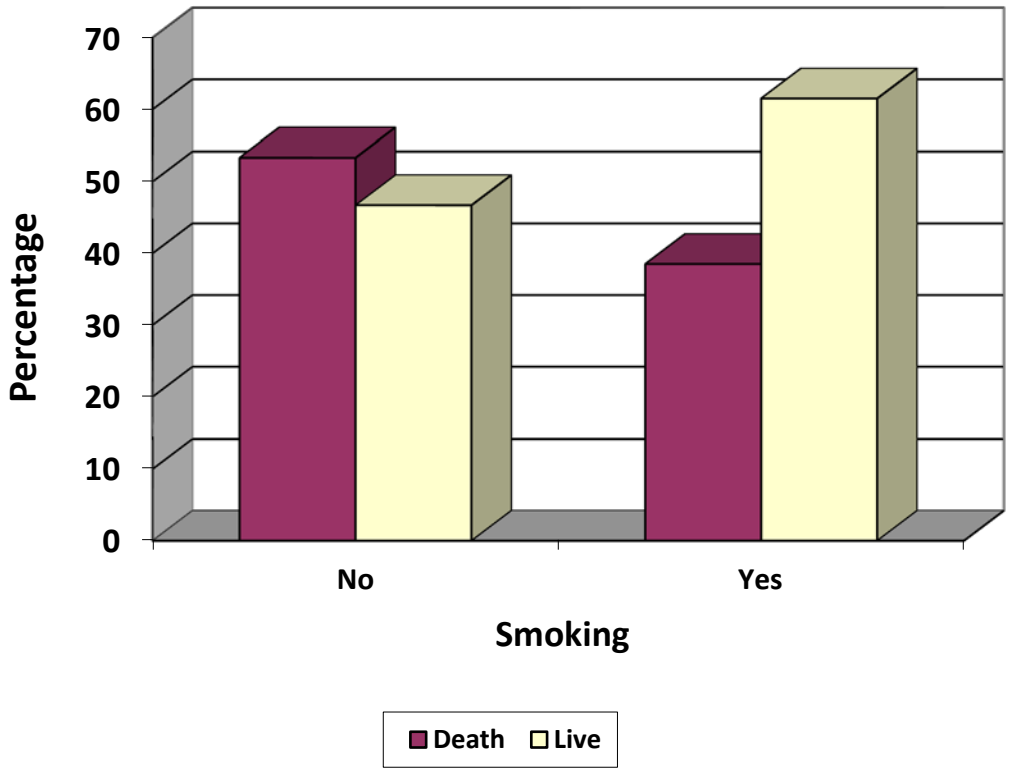
Graph 13: FREQUENCY DISTRIBUTION OF AGE & EJECTION FRACTION:



Tab 20: CHI SQUARE TEST FOR MORTALITY IN SMOKERS:

SMOKING	Death		Live		Total		Statistical inference
	(n=15)	*(100%)*	*(n=65)*	*(100%)*	*(n=80)*	*(100%)*	
No	8	53.3%	25	38.5%	33	41.3%	X^2=1.112 Df=1 .292>0.05 Not Significant
Yes	7	46.7%	40	61.5%	47	58.8%	

Graph 14: CHI SQUARE TEST FOR MORTALITY IN SMOKERS:

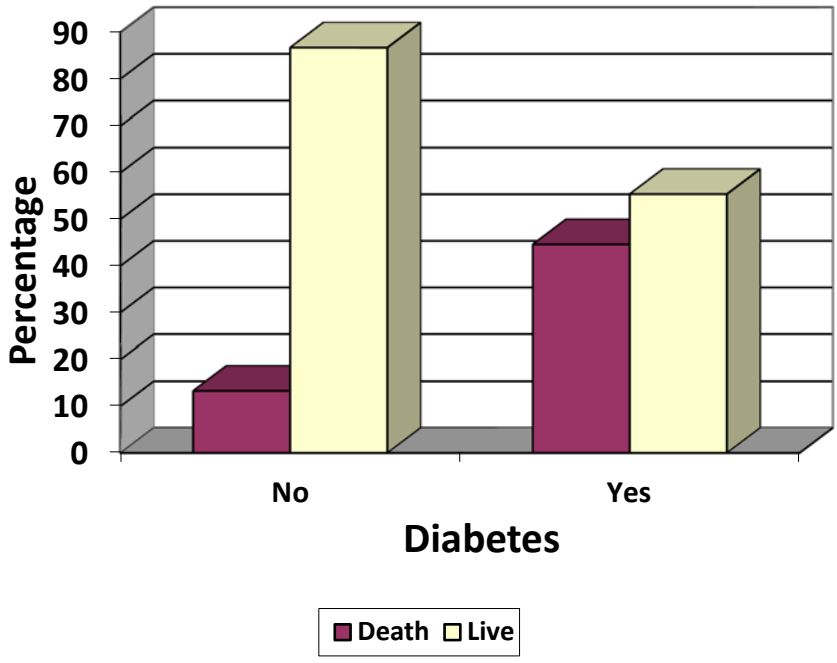


Our study contain 15 members among which smokers were 8 in number (53.3%) and non smokers were (46.7%) and the difference was statistically insignificant.

Tab21: CHI SQUARE TEST FOR MORTALITY IN DIABETES:

DIABETES	**Death**		**Live**		**Total**		**Statistical inference**
	(n=15)	***(100%)***	***(n=65)***	***(100%)***	***(n=80)***	***(100%)***	
No	2	13.3%	29	44.6%	31	38.8%	X^2=5.025 Df=1 .025<0.05 Significant
Yes	13	86.7%	36	55.4%	49	61.3%	

Graph 15 :CHI SQUARE TEST FOR MORTALITY IN DIAB

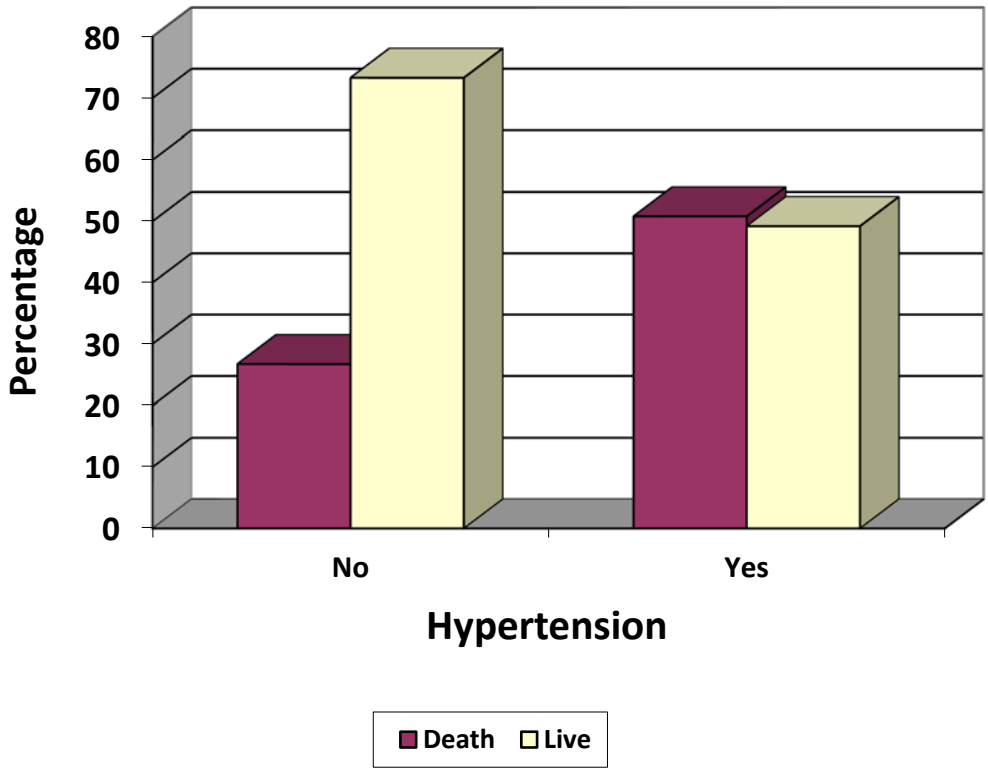


Diabetic persons in the death group were 13 (86.7%) and 2 were non diabetics (13.3%) and the difference was statistically very significant chi square value = 5.025 Df = 1 and p = 0.025(<0.05)

Tab22: CHI SQUARE TEST FOR MORTALITY IN HYPERTENSION:

HYPERTENSION	Death		Live		Total		Statistical inference
	(n=15)	*(100%)*	*(n=65)*	*(100%)*	*(n=80)*	*(100%)*	
No	4	26.7%	33	50.8%	37	46.3%	X^2=2.848 Df=1 .091>0.05 Not Significant
Yes	11	73.3%	32	49.2%	43	53.8%	

Graph 16: CHI SQUARE FOR MORTALITY In HYPERTENSION:



Tab23 : CHI SQUARE TEST FOR MORTALITY IN WBC < 8000

GROUP:

RBS <130 mg%

WBC< 8000 (RBS<130)	**Death**		**Live**		**Total**		**Statistical inference**
	(n=15)	***(100%)***	***(n=65)***	***(100%)***	***(n=80)***	***(100%)***	
No	15	100.0%	50	76.9%	65	81.3%	X^2=4.206 Df=1 .039<0.05 Significant
Yes	0	.0%	15	23.1%	15	18.8%	

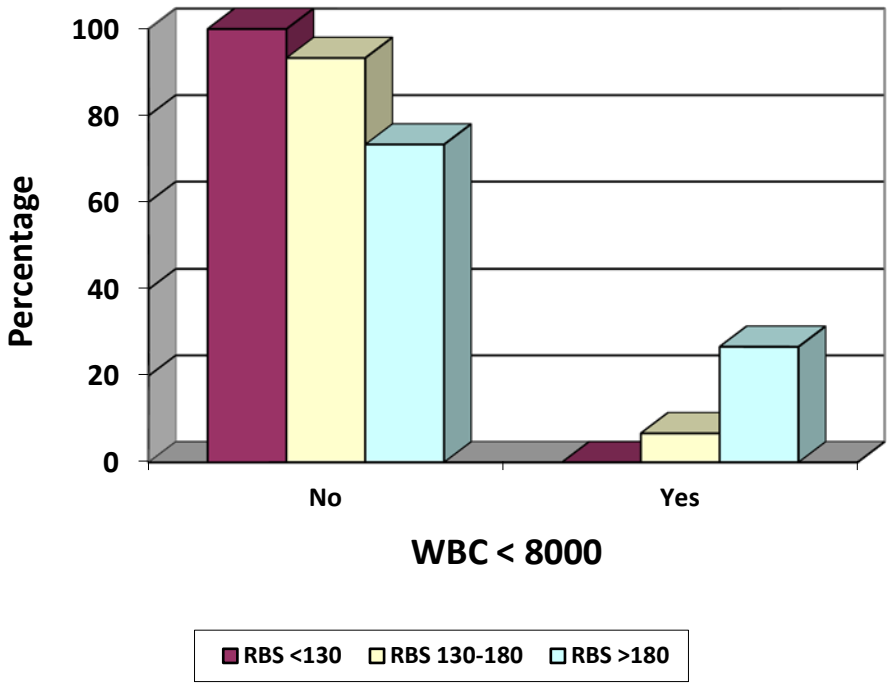
RBS 130 -180 mg%

WBC< 8000 (RBS130-180)	**Death**		**Live**		**Total**		**Statistical inference**
	(n=15)	***(100%)***	***(n=65)***	***(100%)***	***(n=80)***	***(100%)***	
No	14	93.3%	52	80.0%	66	82.5%	X^2=1.501 Df=1 .221>0.05 Not Significant
Yes	1	6.7%	13	20.0%	14	17.5%	

RBS >180 mg%

WBC< 8000-11000 (RBS>180)	Death		Live		Total		Statistical inference
	(n=15)	*(100%)*	*(n=65)*	*(100%)*	*(n=80)*	*(100%)*	
No	11	73.3%	61	93.8%	72	90.0%	X^2=5.698 Df=1 .017<0.05 Significant
Yes	4	26.7%	4	6.2%	8	10.0%	

Graph 17 : CHI SQUARE TEST FOR MORTALITY IN WBC < 8000 GROUP:



Tab 24: CHI SQUARE TEST FOR MORTALITY IN WBC 8000-11000 GROUP:

RBS <130 mg%

WBC: 8000-11000 (RBS<130)	**Death**		**Live**		**Total**		**Statistical inference**
	(n=15)	***(100%)***	***(n=65)***	***(100%)***	***(n=80)***	***(100%)***	
No	15	100.0 %	49	75.4%	64	80.0%	X^2=4.615 Df=1 .032<0.05 Significant
Yes	0	.0%	16	24.6%	16	20.0%	

RBS 130- 180 MG%

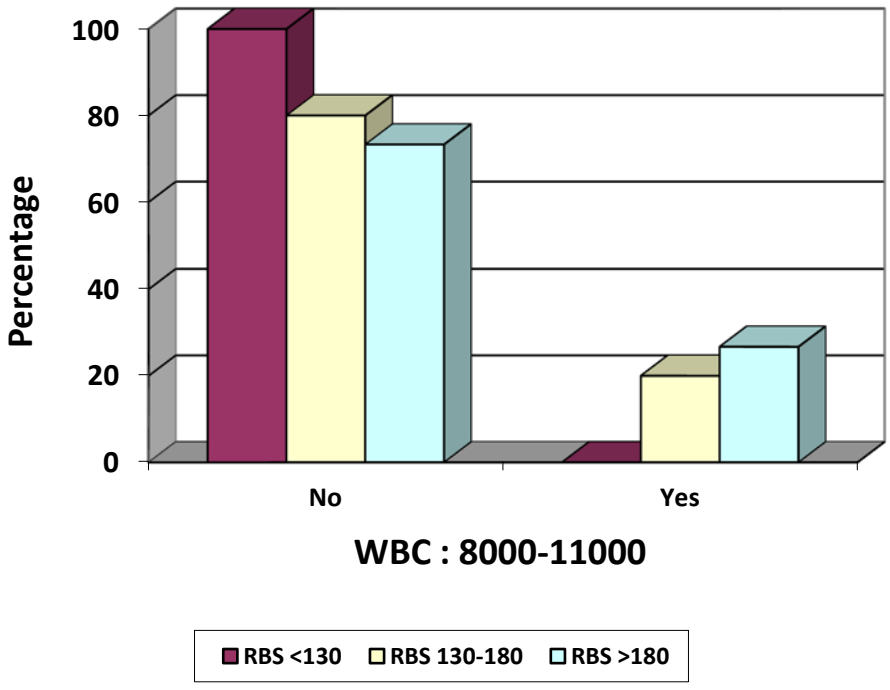
WBC: 8000-11000 (RBS130-180)	**Death**		**Live**		**Total**		**Statistical inference**
	(n=15)	***(100%)***	***(n=65)***	***(100%)***	***(n=80)***	***(100%)***	
No	12	80.0%	56	86.2%	68	85.0%	X^2=.362 Df=1 .547>0.05 Not Significant
Yes	3	20.0%	9	13.8%	12	15.0%	

RBS >180 mg%

	Death		Live		Total		
WBC: 8000-11000 & (RBS>180)	***(n=15)***	***(100%)***	***(n=65)***	***(100%)***	***(n=80)***	***(100%)***	**Statistical inference**
No	12	80.0%	63	96.9%	75	93.8%	X^2=5.957 Df=1 .015<0.05 Significant
Yes	3	20.0%	2	3.1%	5	6.3%	

Graph 18 : CHI SQUARE TEST FOR MORTALITY IN WBC

8000- 11000 GROUP:



Tab 25: CHI SQUARE TEST FOR MORTALITY IN WBC >11000

GROUP:

RBS <130 mg%

WBC> 11000 & (RBS<130)	**Death**		**Live**		**Total**		**Statistical inference**
	(n=15)	***(100%)***	***(n=65)***	***(100%)***	***(n=80)***	***(100%)***	
No	15	100.0%	63	96.9%	78	97.5%	X^2=.473 Df=1 .491>0.05 Not Significant
Yes	0	.0%	2	3.1%	2	2.5%	

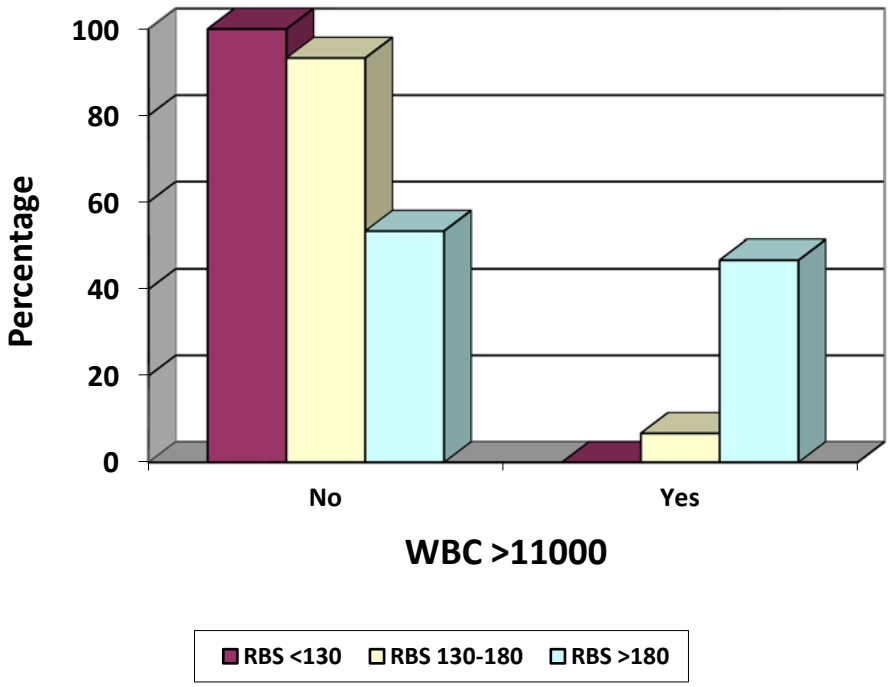
RBS 130-180 mg %

WBC> 11000 & (RBS130-180)	**Death**		**Live**		**Total**		**Statistical inference**
	(n=15)	***(100%)***	***(n=65)***	***(100%)***	***(n=80)***	***(100%)***	
No	14	93.3%	61	93.8%	75	93.8%	X^2=.005 Df=1 .941>0.05 Not signify
Yes	1	6.7%	4	6.2%	5	6.3%	

RBS >180 mg %

WBC> 11000 & (RBS>180)	**Death**		**Live**		**Total**		**Statistical inference**
	(n=15)	***(100%)***	***(n=65)***	***(100%)***	***(n=80)***	***(100%)***	
No	8	53.3%	65	100.0%	73	91.3%	X^2=33.242 Df=1 .000<0.05 Significant
Yes	7	46.7%	0	.0%	7	8.8%	

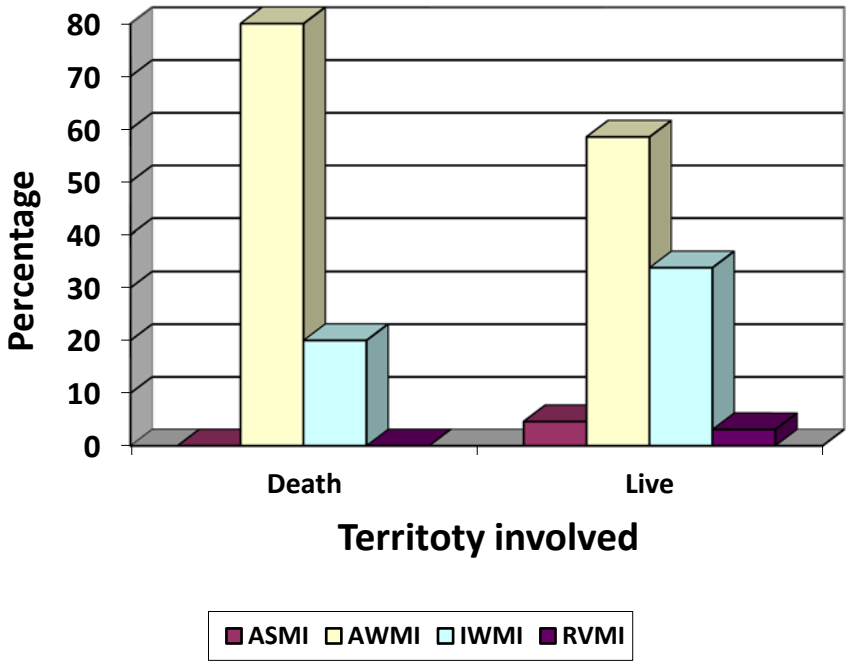
Graph 19: CHI SQUARE TEST FOR MORTALITY IN WBC >11000 GROUP:



Tab 26: CHI SQUARE TEST FOR MORTALITY IN VARIOUS CORONARY TERRITORIES:

TERRITOTY INVOLVED	Death		Live		Total		Statistical inference
	(n=15)	*(100 %)*	*(n=65)*	*(100 %)*	*(n=80)*	*(100 %)*	
ASMI	0	.0%	3	4.6%	3	3.8%	X^2=2.806 Df=3 .422>0.05 Not Significant
AWMI	12	80.0%	38	58.5%	50	62.5%	
IWMI	3	20.0%	22	33.8%	25	31.3%	
RVMI	0	.0%	2	3.1%	2	2.5%	

Graph 20:CHI SQUARE TEST FOR MORTALITY IN VARIOUS CORONARY TERRITORIES:



Tab 27:

Variable	Chi Square	P Value
Killips Class >2	X^2=4.726	P=.000<0.05 Significant
Age	X^2=3.545	P=.471>0.05 Not Significant
WBC Count >11000	X^2=33.242	P=.000<0.05 Significant
RBS >180	X^2=5.698	P=.017<0.05 Significant
Prior IHD	X^2=1.015	P=.314>0.05 Not Significant
Smoker	X^2=1.112	P=.292>0.05 Not Significant
Diabetes mellitus	X^2=5.025	P=.025<0.05 Significant
Sex	X^2=3.242	P=.072>0.05 Not Significant
Hypertension	X^2=2.848	P=.091>0.05 Not Significant
Territory involved	X^2=2.806	P=.422>0.05 Not Significant

Multivariate analysis showed that WBC count and random blood glucose levels are independent predictors for in hospital mortality.

ANALYSIS OF RESULTS:

This study included 80 patients with ST elevation myocardial infarction admitted within 48 hours of symptom onset and their blood sugar values and total WBC counts were compared with the in-hospital mortality.

Our study group comprised of 62 males (77.5%) and 18 females(22.5%). Smokers (n=47) constitute 51.2% of the study group. Patients who were already diabetics and taking treatment were 49 (61.2%). And hypertensives were 43 people(53.7%). The number of patients presenting with killip class 1, 2, 3, 4 were 31(38.8%), 31(31.8%), 14,(17.4%) and 4 (5.0%) respectively. Patients underwent thrombolysis were 54(67.5%) and conservatively managed were26(32.5%).

The patients were stratified into three groups based on their blood sugar values and total white blood cell count.

Blood glucose Group I: Random blood sugar < 130 mg%,

Blood glucose Group II: Random blood sugar 131- 180 mg%, and

Blood glucose Group III: Random blood sugar >I80 mg%

WBC Group I: Total count < 8000 cells/ cu mm.

WBC Group II: Total count 8000-11000/ cu mm and

WBC Group III: Total count >11000 cu/mm.

The total number of deaths during the hospital stay in our study contain 15 members among which smokers were 8 in number (53.3%) and non smokers were (46.7%) and the difference was statistically insignificant. Among the mortality group 9 (60.0%) were men and 6 (40.0%)were women and the sex difference was not statistically significant. Diabetic persons in the death group were 13 (86.7%) and 2 were non diabetics (13.3%) and the difference was statistically very significant chi square value = 5.025 Df = 1 and p = 0.025(<0.05) that shows the diabetic status influences the mortality in our study. Among the 80 patients the mean killip class of recovered patients were 3.0 (SD = 0.756) and that of died people were 1.63(SD = 0.675) and the T value is 6.927 ; Df = 78 ; p = 0.001 that is significant statistically. So the patients admitted with high killip class succumb to the disease.

In the WBC group1 i.e. WBC count <8000/ cu mm. the number of deaths in RBS<130 mg subgroup was 0 ; in RBS 130- 180mg subgroup was 1(6.7%) and in RBS>180mg subgroup was 4(26.7%) and the chi square analysis showed there is a significant association of the last subgroup with the mortality (p = 0.017).

In the WBC group2 i.e. WBC count 8000-11000 cells / cu mm. the number of deaths in RBS<130 mg subgroup was 0 ; in RBS 130- 180mg subgroup was 3(20.0%) and in RBS>180mg subgroup was

4(26.7%) and the chi square analysis showed there is a significant association of the 2^{nd} and 3^{rd} subgroup with the mortality (p = 0.039) and (p = 0.032) respectively.

In the WBC group3 i.e. WBC count >11000/ cu mm. the number of deaths in RBS<130 mg subgroup was 0 ; in RBS 130- 180mg subgroup was 1(6.7%) and in RBS>180mg subgroup was 7(46.7%) and the chi square analysis showed there is a significant association of the last subgroup with the mortality (p = 0.001)

There is also positive correlation between the mean Ejection fraction percentage among the people in these three groups. In the WBC group1 i.e. WBC count <8000/ cu mm the mean ejection fraction in RBS<130 mg subgroup was 51.67% ; in RBS 130- 180mg subgroup was 48.50% and in RBS>180mg subgroup was 42.0%.

Among the mortality group the number of persons in Anterior wall MI, Antero septal MI, Inferior wall MI, Right ventricular MI were 12(80%) , 0(0%), 3(20%), 0(0%) respectively. Analysis showed a p value of 0.422 and hence there is no significant association between the territory involved and the outcome in our study.

DISCUSSION:

In our study we have included 80 cases of acute ST elevation myocardial infarction presented to the hospital within 48 hours of onset of symptoms and got admitted in IMCU/ ICCU of thanjavur medical college hospital, during the period January2014- August 2014.

The patient's history was taken and physical examination findings were noted. At admission patient's random blood glucose values, total WBC count, 12 lead ECG, Echocardiogram were done. The patients were followed during the hospital stay and in-hospital complications like congestive cardiac failure and arrhythmias were noted. Death was the end point of the study.

Admission RBS values, WBC count values , in hospital complications, ejection fraction and in- hospital mortality data were analyzed. Chi- square test and one way ANOVA with post hoc test were used to identify differences between the 3 groups with their subgroups.

The 80 patients that were studied had ST elevation acute myocardial infarction and were divided into three groups 1-3 based on admission (RBS< 130 mg%, RBS 131- 180 mg%, and RBS >I80 mg%. There were 3 subgroups in each group: WBC< 8000 cells/ cu mm, WBC count 8000-11000/ cu mm and WBC count >11000 cu/mm.

.

The mean age of the patients in our study group was 57.83 years. The mean age were compared between these three groups, which were statistically not significant. However previous studies have shown that as age advances there is high rates of poor glycemic status, which may be responsible along with other risk factors.

There were a total of 62 males and 18 females in our study. The mortality rate was compared between the two sex groups which was not significant .But in a previous study by Kahderi et al women had high admission blood glucose compared to males of similar age and have higher mortality.

In our study 43 patients had hypertension and were on treatment, 47 patients gave history of smoking and 44 patients were previously had coronary artery heart diasease and on treatment . There was no statistically significant difference in smokers, hypertensives and prior IHD patients and their mortality. Ranicho et al studied in 2002; an American community-based study showed the presence of high mortality in MI patients with history of smoking, hypertension and recurrent infarction patients. These results are not perfectly matched with ours, and this could be explained by the ethnic and genetic variations between Indian and American people.

The total number of diabetics in our study were 49 persons when we analysed the mortality among these people it was statistically very significant that shows the

diabetic status influences the mortality. Moreover there is a positive correlation between the blood glucose levels and a poor outcome as the patients in high blood glucose group had high killip class and low mean Left ventricular ejection fraction. These results were supported by many previous studies especially the one done by Hafiner et al in which the patients with diabetes have two times high incidence of myocardial infarction and these patients also have four times high mortality compared to controls. Previous studies also showed that congestive cardiac failure is the main complication of diabetics with MI and this may also contribute to the outcome.

In our study the patients with high blood glucose i.e RBS> 180 mg% have significant mortality nearly around 4 times compared to the low blood glucose groups even when adjusted for other risk factors.. These results were comparable to the previous studies done by Qiako et al in which he showed that in non diabetic patients with myocardial infarction the association of 2 hours prandial blood glucose is an independent predictor of poor outcome and future coronary events.

The patients with high WBC count in our study have high mortality around 10 times when compared to the medium and low count groups even when other confounding factors were eliminated. Our results were supported by a study done by Baranni et al . According to his study high wbc count was associated with higher amplitude of ST elevation, high incidence of transmural infarcts, and high

incidence of congestive cardiac failure, ventricular tachycardia and one year mortality rates.

Hence it was found that patients with high WBC count (> 11,000) at admission had a ten fold higher mortality rate than those patients having low WBC count (<8300). Similarly patients with high blood glucose level (>180mg%) had a four fold higher rate of mortality than those patients with low blood glucose(<130) . A multivariate analysis was done of various risk factors like smoking , advanced age, hypertension with mortality and we found that WBC counts and plasma glucose levels were independent risk factors in acute myocardial infarction. These results were in concordance with a study done by Iseikara et al who found out that the patients with a high WBC count had a two fold increase in hospital mortality compared with those with a low W.B.C count and patients with high glucose level had a 2.7 fold increase in mortality compared to the low plasma glucose level. Another study done by Morghan et al described a 9.4 fold higher mortality in patients with high WBC count and high glucose level at admission compared to those with low values.

Several hypotheses were proposed to explain the relation between hyperglycemia and poor outcome in myocardial infarction. Hyperglycemia may be a marker of extensive myocardial damage, reflecting a surge of stress hormones such as catecholamines and cortisol that produce or accentuate an insulin-resistant

state. These changes reduce glucose uptake by the ischemic myocardium and promote lipolysis and increased circulating FFA- free fatty acids. The latter inhibit glucose oxidation by inhibiting aerobic glycolysis and are as such toxic to ischemic myocardium, resulting in increased membrane damage, arrhythmias, and reduced contractility and stroke volume. Alternatively elevated blood glucose levels per se adversely affect outcome through the cumulative effects of several mechanisms, including induction of endothelial dysfunction, oxidative stress, hypercoagulability, and impaired fibrinolysis.

SUMMARY:

- The maximum number of deaths occur in the **age group** of 51- 60 years.The mean age of the patients in our study group was 57.83 years. The mean age were compared between these three groups, which were statistically **not significant.**

- There were a total of 77% males and 23% females in our study. The mortality rate was compared between the two **sex groups** which was **not significant.**

- In our study 43 patients had hypertension , 47 patients gave history of smoking and 44 patients were previously had coronary artery heart diasease . There was **no statistically significant** difference in **smokers, hypertensives and prior IHD** patients and their mortality.

- in our study the mortality among the **diabetic patients** was higher when compared with non diabetics and the association was **statistically significant** that shows the diabetic status influences the mortality. Moreover there is a positive correlation between the blood glucose levels and a poor outcome as the patients in high blood glucose group had high killip class and low mean Left ventricular ejection fraction.

- In our study the patients with **high blood glucose i.e RBS> 180 mg%** have **significant mortality** nearly around 4 times compared to the low blood glucose groups even when adjusted for other risk factors..

- The patients with **high WBC count (>11000)** in our study have **high mortality** around 10 times when compared to the medium and low count groups even when other confounding factors were eliminated.

- A multivariate analysis was done of various risk factors like smoking , advanced age, hypertension with mortality and we found that **WBC counts and plasma glucose** levels were **independent risk factors** in acute myocardial infarction.

LIMITATIONS:

- It is hospital based one step study.
- Limited number of cases were studied.
- Diabetic patients were not excluded from the study.
- Chronic inflammatory conditions causing leukocytosis were not excluded
- Patients were not followed up for future complications after discharge from the hospital.
- Diagnosis of myocardial infarction was based on clinical features and ECG. Cardiac biomarkers were not done.

However the result of the study serve as a baseline for further study of blood sugar levels and WBC count in myocardial infarction and their inclusion in risk scoring systems..

CONCLUSION:

Following conclusions were made after completion of this study:

- Subjects with higher admission RBS(>180 mg%) either diabetic or non diabetic and those with high leukocyte count (>11000) were found to have high incidence of in-hospital mortality.
- Elevated admission RBS and leukocytes levels are associated with increase in cardiac complications and poor cardiac function.
- There was a positive linear correlation between admission RBS and WBC count values with the outcome of the patients.

BIBILIOGRAPHY:

1. Ishihara M , Kojimia S , Sakemato T , Ashada Y , Kinura K et al ;Usefulness Of Combined White Blood Cell Count and Plasma Glucose For Predicting In-Hospital outcomes After Acute Myocardial Infarction . American journal of Cardiol 2006;97:1558-1563.
2. Harrison's Principles Of Internal Medicine volume -2; .18th edn page no:1448-1458.
3. Antman EM,Braunwald ;ST segment elevation Myocardial infarction.In:Zipes, Libby, Bonow, Braunwald editors.Braunwald's Heart disease a textbook of cardiovascular medicine. 7thedn. Philadelphia:Elsiever Saunders;2005.page n0:1141-1142
4. Arthur.C.Guyton, John.E.Hall. Resistance of body to infection -1 In Guyton Textbook of physiology.10th edition.Philadelphia: Elsiever Saunders;2006.p392.
5. Chandler AB, Chapmann I, Erihadt LR, Roberts WC, Schwartz CJ, Sinapius D, et al. Coronary thrombosis in myocardial infarction. Report of a workshop on the role of coronary thrombosis in the pathogenesis of acute myocardial infarction. Am J Cardiol 1974; 34: 823– 833.
6. Cotran ,Ramzi.S. .Disorders of white blood cells In: Kumar Vinay ,Abbas,Fausto editors Robbin's Pathologic basis of disease;7th edn.

.Philadelphia: Elsiever Saunders;2005.page no: 9-10

7. R. Ross, Atherosclerosis—an inflammatory disease.N Engl J Med 1999; 340:115– 126.
8. Amaro, J.R. Gonzalez-Juanatey and C Iglesias et al., Leukocyte count as a predictor of the severity ischaemic heart disease as evaluated by coronary angiography .Rev Port Cardiol 1993;12:913–917
9. R. Hajji-Ali, W. Zereba, R. Ezedhine and A.J. Mossy, Relation of the leukocyte count to recurrent cardiac events in stable patients after acute myocardial infarction, American Journal of Cardiology 2001;88:1221–1224.
10. C.P. Canon, C.H. McCabbe, R.G. Willcox, J.H. Bentley, E. Braunwald and the OPUS-TIMI 16 Investigators, Association of white blood cell count with increased mortality in acute myocardial infarction and unstable angina pectoris;American Journal of Cardiology 2001; 87:636–639.
11. H.V. Barron, C.P. Cannon, S.A. Murphy, E. Braunwald and C.M. Gibson, Association between white blood cell count, epicardial blood flow, myocardial perfusion, and clinical outcomes in the setting of acute myocardial infarction: a Thrombolysis In Myocardial Infarction 10 substudy, Circulation 2000; 102:2329– 2334.

12. P. Reizenstein, The haematological stress syndrome, British Journal of Haematology 1979;43:329– 334.
13. Goyal A, Mahaffey K, Garg J, Nicolau JC, Hochman JS, Weaver WD, Theroux P, Oliveira GBF, Todaro TG, Mojcik CF, Armstrong PW, Granger CB. Prognostic significance of the change in glucose level in the first 24h after acute myocardial infarction: results from the CARDINAL study.EurHeartJ.2006;27:1289 –1297.
14. Beckman, J.A., M.A. Creager, and P. Libby, Diabetes and atherosclerosis: epidemiology, pathophysiology, and management. Jama, 2002. 287(19): p. 2570-81.
15. Drivsholm, T., et al., Increasing prevalence of diabetes mellitus and impaired glucose tolerance among 60-year-old Danes. Diabet Med, 2001. 18(2): p. 126-132.
16. Norhammar A, Malmberg K, Ryden L, et al for the register of Information and knowledge about Swedish Heart Intensive Care Admissions (RIKS-HIA). Under utilisation of evidence-based treatment partially explains for the unfavourable prognosis in diabetic patients with acute myocardial infarction.European Heart Journal 2003;24:838-844.

17. Norhamear A, Teneriz A, Nilson G, et al. Glucose metabolism in patients with acute myocardial infarction and no previous diagnosis of diabetes mellitus: a prospective study. Lancet 2002;359:2140-2144.

18. Hindman L, Falko JM, Lalonde M, et al. Clinical profile and outcomes of diabetic and nondiabetic patients in cardiac rehabilitation. Am Heart J 2005;150:1046-1051.

19. Malmberg K, Yusuf S, Gerstein HC, et al. Impact of diabetes on long-term prognosis in patients with unstable angina and non-Q-wave myocardial infarction: results of the OASIS (Organization to Assess Strategies for Ischemic Syndromes) registry. Circulation 2000;102:1014-1019.

20. Rachmani R, Slavachevski I, Berla M, et al. Teaching and motivationg patients to control their risk factors retards progression of cardiovascular as well as microvascular sequelae of type 2 diabetes mellitus – a randomized prospective 8 years follow-up study. Diabetic Medicine 2005;22:410-414.

21. van der Horst IC, Zijlstra F, van't Hof AW, et al. Glucose-insulin-potassium infusion in patients treated with primary angioplasty for acute myocardial infarction: the glucose-insulin-potassium study (GIPS-1): randomized trial. J Am Coll Cardiol 2003;42:784-791.

23. Malmberg K, Ryden L, Wedel H, et al for the DIGAMI 2 Investigators. Intense metabolic control by means of
insulin in patients with diabetes mellitus and acute myocardial infarction (DIGAMI 2): effects on mortality
and morbidity. Eur Heart J 2005;26:650-661.

24. Apstein CS. Increased glycolytic substrate protection improves ischemic cardiac dysfunction
and reduces injury. Am Heart J 2000;139:S107-S114.

25. Legtenberg RJ, Houston RJ, Oeseburg B, Smits P. Physiological insulin concentrations
protect against ischemia-induced loss of cardiac function in rats. Comp Biochem Physiol A
Mol Integr Physiol 2002;132:161-67.

26. Capes SE, Hunt D, Malmberg K, Gerstein HC. Stress hyperglycaemia and increased risk of
death after myocardial infarction in patients with and without diabetes: a systematic
overview. Lancet 2000;355:773-78.

27.Lazar HL, Chipkin SR, Fitzgerald CA, Bao Y, Cabral H, Apstein CS. Tight Glycemic Control in Diabetic Coronary Artery Bypass Graft Patients Improves Perioperative Outcomes and Decreases Recurrent Ischemic Events. Circulation 2004; 109:1497-502.

28.Sack MN, Yellon DM. Insulin therapy as an adjunct to reperfusion after acute coronary ischemia: a proposed direct myocardial cell survival effect independent of metabolic modulation. J Am Coll Cardiol 2003;41:1404-7.

29. Stubbs P, Laycock J, Alaghband-Zadeh J, et al. Circulating stress hormone and insulin concentrations in acute coronary syndromes:identification of insulin resistance on admission. Clin Sci 1999;96: 589-95.

30. Capes S, Hunt D, Malmberg K, et al. Stress hyperglycaemia and increased risk of death after myocardial infarction in patients with and without diabetes: a systematic overview. Lancet 2000; 355: 773-8.

31. Cao J, Hudson M, Jankowski M, et al. Relation of chronic and acute glycemic control on mortality in acute myocardial infarction with diabetes mellitus. Am J Cardiol 2005; 96: 183-6.

32. Bolk J, van der Ploeg T, Cornel J, et al. Impaired glucose metabolism predicts mortality after a myocardial infarction. Int J Cardiol 2001; 79: 207-14.

33.Gross E, LaDisa J Jr, Weihrauch D, et al. Reactive oxygen species modulate coronary wall shear stress and endothelial function

during hyperglycemia. Am J Physiol Heart Circ Physiol 2003; 284: 1552-9.

34. Das U. Free radicals, cytokines and nitric oxide in cardiac failure and myocardial infarction. Mol Cell Biochem 2000; 215: 145-52.

40. Oliver M, Opie L. Effects of glucose and fatty acids on myocardial ischaemia and arrhythmias. Lancet 1994; 343: 155-8.

35. Holubarsch C, Ruf T, Goldstein D, et al. Existence of the Frank-Starling mechanism in the failing human heart: investigations on the organ, tissue, and sarcomere levels. Circulation 1996; 94: 683-9.

36. Shen X, Jia S, Li H. The influence of admission glucose on epicardial and microvascular flow after primary angioplasty. Chin Med J 2006; 119: 95-102.

37. Timmer J, Ottervanger J, de Boer M, et al. Hyperglycemia is an important predictor of impaired coronary flow before reperfusion therapy in ST-segment elevation myocardial infarction. J Am Coll Cardiol 2005; 45: 999-1002.

38. Kosuge M, Kimura K, Ishikawa T, et al. Persistent hyperglycemia is associated with left ventricular dysfunction in patients with acute myocardial infarction. Circ J 2005; 69: 23-8.

39. Farsi A, Domeneghetti MP, Fedi S, et al. High prevalence of anti-beta 2glycoprotein I antibodies in patients with ischemic heart disease. Autoimmunity 1999;30(2):93±8.

40.Marx N, Neumann FJ, Ott I, et al. Induction of cytokine expression in leukocytes in acute myocardial infarction. J Am Coll Cardiol 1997;30:165±70

41. Frangogiannis NG, Mendoza LH, Lindsey ML, et al. IL-10 is induced in the reperfused myocardium and may modulate the reaction to injury. J Immunol 2000;165(5):2798±808.

42. Blum A, Sclarovsky S, Rehavia E, Shohat B. Levels of T-lymphocyte subpopulations, interleukin-1b, and soluble interleukin-2 receptor in acute myocardial infarction. Am Heart J 1994;127:1226±30 .

43. Murohara T, Kamijikkoku S, Honda T. Increased circulating soluble intercellular adhesion molecule-1 in acute myocardial infarction: possible predictor of reperfusion ventricular arrhythmias. Crit Care Med 2000;28(6):1861±4.

44. Hojo Y, Ikeda U, Zhu Y, et al. Expression of vascular endothelial growth factor in patients with acute myocardial infarction. J Am Coll Cardiol 2000;35(4):968±73

PROFORMA

NAME: IP. NO:

AGE: DOA:

SEX: DOD:

OCCUPATION: ADDRESS:

MARITAL STATUS:

STATUS AT DISCHARGE:

PRESENTING COMPLAINTS:

I. HISTORY OF PRESENTING ILLNESS:

A. CHEST PAIN:

Site: Precordial/ Restrosternal/Epigastric/ Shoulder/ Neck

Duration:

Nature: Squeezing/Crushing/Compressive/Tightness

Radiation: Arm/ Back/ Epigastric/ Neck

Frequency:

Aggravating Factor:

Relieving Factor:

Associated :sweating/ vomiting/palpitations/ breathlessness

B. BREATHLESSNESS:

Onset: Sudden/ Gradual Grade: I/II/III/IV

Orthopnea: Yes/ No

PND: Yes/No

C. COUGH

Productive/ Non Productive

Haemoptysis: Yes/ NO

D.SYNCOPAL ATTACKS: Yes/ No

E. SWELLING OF LEGS/ FACE: Yes /No

F. OLIGURIA : Yes /No

G. CONVULSIONS: Yes /No

H. OTHER SYMPTOMS:

II. PAST HISTORY

HISTORY	PRESENT/ABSENT	DURATION	TREATMENT
IHD- Angina			
Infarction			
Diabetes			
Hypertension			
RHD			
Syphilis			
TIA/ Stroke			

III. PERSONAL HISTORY

1. Diet: Vegetarian/ Mixed

2. Bladder: Normal/ Polyuria/ Anuria/Dysuria

5. Bowel: Normal/ Constipated /Loose stools

6. Menstrual history :Normal/Irregular/ Postmenopausal

7. Exposure to STD: Present/ Absent

8. Habits Duration Type Quantity

Smoking :

Alcohol:

Tobacco Chewing :

IV. GENERAL PHYSICAL EXAMINATION

1) Built Well/Moderate Poor

2) Nourishment Obese/Average Poor

3) Emotional state Calm/Anxious Restless

4) Pallor Present/ Absent

5) Cyanosis Present/ Absent

6) Icterus Present/ Absent

7) Clubbing Present/ Absent

8) Pedal edema Present/ Absent

9) Lymphadenopathy Present/ Absent

10) Extremities Warm/ Cold

103

V. VITAL SIGNS

Pulse

Blood pressure

Respiratory rate

Temperature

VI. SYSTEMIC EXAMINATION

CVS EXAMINATION

1) Pulse

Rate

Rhythm

Volume

Character

Condition of Vessel Wall

Radio Femoral Delay

2) JVP –Normal /Raised

A. INSPECTION

Precordium Normal/Bulged

Apical impulse Visible / Non Visible

Other pulsation

B. PALPATION

Apical impulse Location, Character

Palpable Heart Sounds

Thrills Apex

Parasternal area

Any other

104

C. PERCUSSION

Cardiomegaly

Pericardial effusion

D. AUSCULTATION

Heart sounds

S3/S4 Present/ Absent

Murmur Timing/Location/Character/Radiation/Grade

Pericardial rub

Basal crepitations

Others

KILLIP CLASS:

RESPIRATORY SYSTEM:

PER ABDOMEN:

CENTRAL NERVOUS SYSTEM

105

INVESTIGATIONS

I. BLOOD

HAEMOGLOBIN gm/dl

TC Cells/mm3

DC

NEUTROPHILS %

LYMPHOCYTES %

EOSINOPHILS %

BASOPHILS %

MONOCYTES %

ESR mm/1hr

CARDIAC ENZYMES : CPK-MB IU/L

II.URINE

ALBUMIN

SUGAR

MICROSCOPY

III.BIOCHEMISTRY

RBS mg/dl

TOTAL CHOLESTEROL mg/dl

HDL CHOLESTEROL mg/dl

LDL CHOLESTEROL mg/dl

VLDL CHOLESTEROL mg/dl

TRIGLYCERIDE mg/dl

106

IV.ELECTROCARDIOGRAPHY

V.ECHOCARDIOGRAPHY

EJECTION FRACTION

CONCLUSIONS

OTHER RELEVANT INVESTIGATIONS

DIAGNOSIS

THROMBOLYSIS - DONE/NOT DONE

IN HOSPITAL COMPLICATIONS

CCF/LVF

Cardiogenic shock

Arrythmias

Thromboembolism

Precarditis

Rupture of Interventricular septum

Rupture of papillary muscle

Aneurysm

Any other

FOLLOW UP UPTO TO DATE OF DISCHARGE

LIST OF ABBREVIATIONS

AMI– Acute myocardial infarction

AF– Atrial Fibrillation

CAD – Coronary Artery Disease

CCF – Congestive Cardiac Failure

CHD – Coronary Heart Disease

CK-MB – Creatinine Kinase-MB

CVD – Cardiovascular diseases

ECG – Electrocardiogram

IHD – Ischemic Heart Disease

LVF – Left ventricular Failure

MR – Mitral Regurgitation

MI – Myocardial infarction

RBS – Random Blood Sugar

SA– Sinoatrial node

STEMI – ST Elevation myocardial infarction

VT – Ventricular Tachycardia

WBC – White Blood Cell

MASTER CHART

S.NO	IP. NO	AGE	SEX	SMOKING	DM	HT	PRIOR IHD	KILLIP CLASS	THROMBOLYSIS	WBO< 8000			WBC 8000-11000			WBC > 11000			TERRITOTY INVOLVED	EJECTION FRACTION	OUTCOME D/R
										RBS <130	130-180	RBS >180	RBS < 130	130-180	RBS >180	RBS < 130	130-180	RBS >180			
1	1490527	34	M	Y	N	Y	Y	1	+	Y									AWMI	50	R
2	1489271	80	F	N	N	Y	N	2	+		Y								IWMI	50	R
3	1487224	36	M	Y	N	N	N	2	-					Y					AWMI	52	R
4	1488312	50	M	M	Y	Y	Y	1	-	Y									AWMI	50	R
5	1484857	37	M	Y	N	Y	N	2	+					Y					ASMI	52	R
6	1486695	60	M	Y	Y	N	N	3	-			Y							ASMI	55	R
7	1486421	70	F	N	Y	N	N	3	+						Y			Y	AWMI	42	D
8	1485091	50	F	N	Y	N	Y	1	+				Y						IWMI	51	R
9	1482551	74	M	Y	N	N	Y	2	-		Y								IWMI	48	R
10	148864	60	F	N	Y	Y	N	4	-						Y				RVMI	45	D
11	3400	60	F	N	Y	Y	N	1	+				Y						JWMI	50	R
12	8611	65	F	N	Y	Y	Y	2	+			Y							AWMI	38	D
13	3425	66	M	Y	Y	Y	N	1	+	Y									IWMI	52	R
14	3365	53	M	Y	N	N	Y	2	-								Y		ASMI	58	R
15	2824	54	M	Y	N	N	N	1	+				Y						AWMI	50	R
16	842	72	M	N	N	Y	N	1	-	Y									AWMI	54	R
17	1200	42	M	N	N	N	N	2	+					Y					JWMI	55	R

18	671	50	M	Y	Y	Y	Y	4	+									Y	AW MI	32	D
19	1492232	56	M	Y	N	N	Y	3	+						Y				AW MI	48	R
20	1490197	65	M	N	Y	N	N	2	+					Y					AW MI	51	R
21	7211	85	M	N	Y	N	Y	2	-								Y		IW MI	50	R
22	7079	50	M	N	N	N	Y	1	+				Y						AW MI	54	R
23	6584	60	F	N	Y	Y	Y	2	+		Y								IW MI	50	R
24	5588	66	M	Y	Y	Y	Y	3	+					Y					IW MI	29	D
25	5381	55	M	Y	N	N	N	1	+				Y						AW MI	54	R
26	5322	28	M	Y	N	N	N	1	-	Y									AW MI	55	R
27	5216	60	F	N	Y	Y	N	3	+			Y							AW MI	30	D
28	4863	75	M	N	Y	Y	Y	2	-		Y								AW MI	38	R
29	4378	60	M	Y	Y	Y	N	3	-					Y					AW MI	33	D
30	4150	45	M	Y	N	N	N	2	+		Y								IW MI	48	R
31	10397	56	M	Y	N	Y	Y	2	+		Y								AW MI	42	R
32	9791	55	M	N	N	N	N	3	+			Y							IW MI	40	R
33	9782	40	M	Y	Y	Y	Y	1	+				Y						IW MI	50	R
34	9006	53	F	Y	Y	N	N	1	-	Y									IW MI	52	R
35	8445	60	M	Y	Y	N	Y	2	+					Y					IW MI	54	R
36	8677	46	M	N	N	Y	Y	2	+					Y					AW MI	53	R
37	8025	54	M	N	N	Y	Y	3	-									Y	AW MI	40	D
38	7325	47	M	N	N	N	N	2	+		Y								AW MI	55	R
39	7319	63	M	Y	Y	N	N	1	+				Y						AW MI	52	R
40	7470	60	M	Y	N	Y	N	1	-	Y									IW MI	50	R

41	14527	62	M	Y	Y	N	Y	1	+							Y			AW MI	49	R
42	14292	50	M	Y	Y	N	Y	1	+				Y						AW MI	52	R
43	13335	60	M	Y	Y	N	Y	2	-		Y							Y	AW MI	38	D
44	13076	72	M	Y	N	Y	Y	2	+				Y						AW MI	53	R
45	12947	50	M	Y	Y	Y	N	1	-	Y									IW MI	55	R
46	11653	68	M	Y	N	N	N	3	+						Y				IW MI	48	R
47	12040	45	M	Y	Y	Y	Y	1	+	Y									AW MI	53	R
48	11259	63	M	Y	Y	Y	Y	3	+			Y						Y	AW MI	45	D
49	10499	56	M	Y	N	Y	Y	1	+				Y						AW MI	55	R
50	10449	48	M	Y	Y	Y	N	2	-		Y								IW MI	50	R
51	20101	37	M	Y	N	Y	N	1	-	Y									AW MI	50	R
52	20797	65	M	Y	Y	Y	Y	2	+		Y								AW MI	48	R
53	20155	70	F	N	Y	Y	Y	2	+					Y				Y	AW MI	50	D
54	17671	47	F	N	N	N	Y	1	+				Y						IW MI	55	R
55	18558	58	M	Y	Y	N	N	3	+								Y		AW MI	48	R
56	18236	65	M	Y	Y	N	Y	1	+				Y						AW MI	53	R
57	16903	55	M	N	N	N	Y	3	-			Y							AW MI	42	D
58	15651	61	F	N	Y	Y	Y	2	+		Y								RV MI	50	R
59	14582	62	M	N	N	N	Y	1	+				Y						AW MI	55	R
60	14506	35	M	Y	Y	Y	N	2	-								Y		AW MI	51	R
61	28355	80	F	N	Y	N	Y	1	+							Y			AW MI	50	R
62	28150	69	F	N	Y	N	Y	2	+		Y								AW MI	52	R
63	25264	55	M	Y	Y	Y	Y	2	+					Y					AW MI	55	R

64	25780	76	M	Y	Y	N	Y	4	-								Y		AW MI	30	D
65	24792	59	M	Y	N	Y	N	1	+	Y									AW MI	49	R
66	24453	75	M	Y	N	Y	N	1	+				Y						AW MI	51	R
67	20986	56	M	N	Y	Y	N	1	+	Y									IW MI	50	R
68	22175	67	M	Y	Y	Y	Y	4	-									Y	AW MI	34	D
69	21912	52	F	N	Y	N	N	2	+		Y								AW MI	55	R
70	21260	65	M	Y	Y	Y	Y	3	-			Y							AW MI	45	R
71	31158	70	M	N	Y	N	Y	1	-	Y									AW MI	50	R
72	35661	65	M	N	Y	Y	N	2	+				Y						AW MI	52	R
73	35875	53	M	Y	N	Y	Y	2	+		Y								AW MI	55	R
74	35097	60	F	N	Y	Y	N	1	-				Y						AW MI	53	R
75	33697	57	M	Y	Y	N	Y	1	+	Y									IW MI	50	R
76	33232	62	M	Y	N	N	N	2	+					Y					IW MI	48	R
77	32714	49	F	N	Y	Y	N	2	-						Y				IW MI	48	D
78	30922	55	M	Y	Y	Y	Y	3	+			Y							IW MI	45	R
79	30102	65	F	N	Y	N	Y	2	+					Y					AW MI	52	R
80	28618	55	M	Y	Y	Y	Y	1	+	Y									IW MI	55	R

KEY :

Y	YES
N	NO
F	FEMALE
M	MALE
D	DEATH
R	RECOVERY
+	THROMOLYSIS DONE
-	THROMOLYSIS NOT DONE
ASMI	ANTEROSEPTAL MYOCORDIAL INFARCTION
AWMI	ANTERIOR WALL MYOCORDIAL INFARCTION
IWMI	INFERIOR WALL MYOCORDIAL INFARCTION
RVMI	RVMI RIGHT VENTRICULAR MYOCARDIAL INFARCTION

CONSENT FORM

I _____ hereby give consent to participate in the study conducted by **DR .R.VINOTH KANNAN** Post graduate in the Department of General Medicine ,Thanjavur Medical College & Hospital, Thanjavur – 613004 and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease. I also give consent for further investigations

Place :

Date : Signature of participant

INFORMATION SHEET

- We are conducting a prospective study on **"A STUDY ON PROGNOSTIC SIGNIFICANCE OF WHITE BLOOD CELL COUNT AND BLOOD GLUCOSE LEVELS AT ADMISSION IN ST ELEVATION MYOCARDIAL INFARCTION"** in the Department of General Medicine , Thanjavur Medical College & Hospital, Thanjavur – 613004.

- At the time of announcing the results and suggestions, name and identity of the patients will be confidential.

- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator **Signature of participant**

Date: